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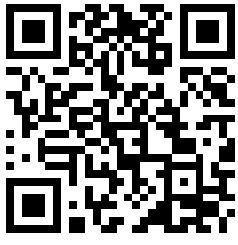

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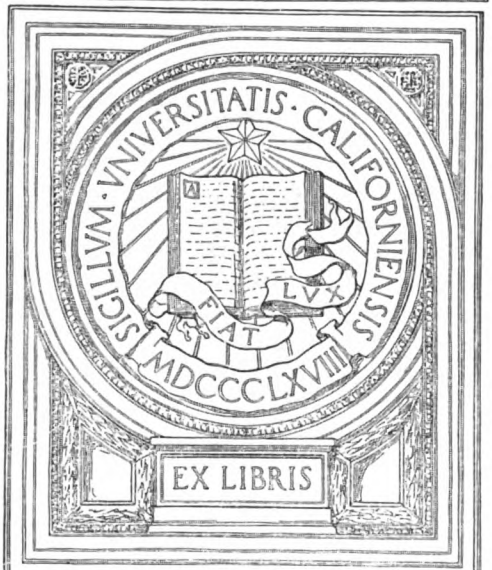

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THE

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A LECTURE
ON
CHOREA.

Delivered at the Hospital for Sick Children, Great Ormond Street, W.C.

By A. E. GARROD, M.A., M.D., F.R.C.P., Senior Physician to the Hospital.

LADIES AND GENTLEMEN,—What is known as chorea is, in a sense, a disease within a disease. It is no mere isolated symptom; it is much more than a variety of inco-ordinate movement. It is a group of symptoms; or a symptom-complex. In one case one symptom, and in another case another symptom assumes the chief prominence, but they all occur in so close association that one can hardly doubt that they owe their origin to some definite morbid event, and that morbid event is a manifestation of rheumatism. At one time there used to be much discussion as to the relationship, if any, between chorea and rheumatism, but such discussions are now things of the past. They turned very largely upon differences in the meanings attached to terms by those who took part in the discussion. If one regards rheumatism as a disease of the joints which is apt to be complicated by lesions of the heart and other organs, it is obvious that chorea is not rheumatism. If, on the other hand, one regards rheumatism as a general infective process or disease, of which the heart troubles and the joint troubles are alike manifestations, then it is equally obvious that chorea is rheumatism. However, into this point of the relationship of chorea and rheumatism I do not propose to enter at any length to-day, believing that the time at our disposal can be better occupied. But one may mention that even that variety of chorea which is least obviously rheumatic, namely, the chorea of pregnancy, is now known to be usually rheumatic. This has been very clearly brought out in a paper

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by Sir Dyce Duckworth, which was published in the 'St. Bartholomew's Hospital Reports' two or three years ago. In that investigation he took a series of cases of the chorea of pregnancy and showed that the patients presented various other rheumatic manifestations.

I said just now that chorea was a group of symptoms, and these symptoms fall into certain definite sub-groups. First of all we will consider the motor phenomena, by which I mean the inco-ordinate movements. These are the most obvious phenomena of chorea. At the same time they are, in all vital respects, the least important of them all. This is one of many instances in which the most obvious features of a morbid condition are not necessarily the most important. In the second sub-group one might put the emotional and mental phenomena, and emotional phenomena and mental phenomena are not quite the same thing. Still it is so difficult to draw the line between them that they are better put into one sub-group. Thirdly, there are the paralytic symptoms. Those sub-groups include all the true phenomena of chorea as such. Associated with chorea there are very apt to occur — seeing that chorea is a form of rheumatism—other rheumatic phenomena, such as cardiac troubles, articular troubles, nodules and the like. They are not part of the chorea, but they occur in close association with it. It is the first three groups, the motor, the emotional, and the paralytic, which constitute chorea, and those are the symptoms which hang together so closely and occur in such intimate association that one cannot doubt that they are due to one single morbid event. What the nature of that morbid event is we do not yet know. There is a good deal to be said in favour of its being a local brain lesion; notably the tendency to a hemiplegic distribution of the motor trouble, and also the observations which have been made on the presence of micro-organisms in different regions of the brain in connection with chorea. On the other hand, there are some features which suggest that it may be a toxic phenomenon due to the formation of some definite bacterial toxin, such as that which in diphtheria produces diphtheritic paralysis, or that which in tetanus produces the nervous phenomena which are the most characteristic features of that disease. Of all morbid conditions chorea is probably the easiest to diagnose, and in the great majority of cases no mistake is possible. The age of the patient affords a clue to a certain extent, because chorea is essentially a phenomenon of childhood, and its age limits correspond very closely to those in which rheumatic nodules appear. There is nothing remarkable in some manifestations of a systemic disease being confined to, or being commoner in, childhood that in adult life. The same is seen in connection with other infective processes. Tubercle takes on different aspects in children to those which it usually assumes in the adult; witness the great frequency of tubercular meningitis as compared with its rarity in adults, and, again, the much greater frequency of tubercular peritonitis in childhood. The pneumococcal disease behaves in the same way. Empyema is especially common in children, to mention only one of the pneumococcal manifestations, and in young children, a fact you are all familiar with, the lung trouble due to pneumococcus takes the form of broncho-pneumonia very much more frequently than that of lobar pneumonia, whereas in adult life lobar pneumonia is the common form.

The movements of chorea are essentially inco-ordinate, and they shift from limb to limb. The same movement is seldom repeated, at any rate more than once or twice in succession, and in that respect choreic movements tend to differ from others with which they might otherwise be liable to be confused, especially with what has been called habit chorea, but which is very much better termed habit spasm. A variety of different morbid conditions may lead to the development of movements somewhat resembling those of chorea. Not very long ago a child was admitted into this hospital with the diagnosis of chorea, and it turned out that the trouble was tubercular meningitis. Again in adult life one sees such conditions as the so-called post-hemiplegic chorea, and a variety of other conditions more or less simulating true chorea. They are not very liable to be mistaken for it, except in a comparatively limited number of cases. The violence of the movements, as also their range, varies immensely in different cases of chorea. In extent also they vary; they may be bilateral, affecting all the limbs and all parts of the body at once. Rarely they are strictly hemiplegic. More often they are more pronounced on one side than on the other. They may be so slight as scarcely to attract any attention, and in a certain

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number of children admitted on account of rheumatism one sees, if one watches the patient, a twitch or a twist which reveals the presence of a very slight form of chorea, but which may not have attracted attention previously. Such slight choreic movements may be overlooked unless carefully watched for. On the other hand the movements may be so violent that the patient requires to be put into a padded bed, and tends to bruise and graze himself to a not unimportant extent. All the voluntary muscles are liable to be seats of the movements of chorea, those of the face and neck as well as those of the limbs. The respiratory muscles are usually affected. It would seem that the involuntary muscles also have their share in the motor disturbance. Certainly an irregularity of the heart's action is a very common phenomenon in chorea. It is a simple irregularity; it may be very trifling, or it may be obvious on a most casual feeling of the pulse. Even the pupils seem to be affected to some extent, as they are erratic in their movements: in contracting they may appear to struggle, and after contracting a little way may revert towards their previous position and eventually obey the stimulus, suggesting that their muscular structures also are not immune from the motor disturbance which is going on in chorea. It is not often the case that the violent movements are a source of any danger to the patient. It is perfectly true that there are cases which are required to be admitted to hospital because of the violence of the movements, because of the inability of the child to walk or to stand, or to be controlled and so on, but these are the least dangerous of the phenomena associated with the disease, and I think one is justified in saying that they are in many respects the least important. Mental and emotional manifestations are never wholly absent from a case of chorea. In some very slight cases they may require to be looked for, but in a considerable number of the cases they are prominent and obvious, and in a certain limited number of cases they dominate the whole clinical picture. They are of many different kinds and are seen in various degrees. The slighter degrees of emotional disturbance are among the commonest features. If one approaches the bed of a choreic patient the child often bursts into tears, or smiles unmeaningly and unnecessarily. High spirits or low spirits may occur in different cases, or they may alternate in the same case. Very little is sufficient to produce considerable emotional disturbance. We all know what a large part in the causation of the disease has been assigned to such influences as fright and strain at school and the like; we also know how little it takes to frighten a choreic child, and personally it is my firm conviction that the fright and the strain are rather symptoms than causes of chorea. In investigating this subject one has to rely upon the statements of the child's parents, and in some cases one can by careful inquiry make out that some symptoms were noticed before the occurrence of the fright. It may have been noticed that the child dropped things, or that it seemed fidgety, or was abnormal or unusual in some slight respect. Again, inquiry will often show that the fright which was supposed to have caused the disease was really caused by some quite inadequate event. The amount of emotional disturbance which results is such as you would expect at the onset of chorea, but not such as would be expected in a normally healthy child exposed to the same frightening case. I do not wish to labour this point, but I think it is one of very great interest, and believe that careful inquiry would confirm the fact that the fright and perhaps the strain at school are symptoms of the pre-choreic stage, or had occurred actually in the choreic condition, rather than that the fright was a real cause of the onset of the symptoms. An alternative explanation would be that the child being in a condition, owing to the rheumatic infection, to acquire chorea, the form in which the infection manifests itself is determined by such external causes as fright, or by overwork at school and so on. The emotional disturbance in a case may be so great that it is not advisable to disturb the child at all, or even to carry out any complete examination. Therefore you may conclude that in some cases it is not at all desirable to give any clinical demonstration at the bedside of the patient. In bad cases of chorea one occasionally sees a state of almost complete apathy. The child's face looks like a mask, the child does not betray any emotion, and, indeed, it seems to have no thoughts behind its features. The speech, which in the early stages in the time of chief motor disturbance was rendered difficult by the inco-ordinate movements of the parts concerned in articulation, may cease entirely from quite a

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different cause. We are dealing in such cases with a species of aphasia, with inability to bring the mechanism of speech into work, and for weeks together the child may lie speechless, presenting the clinical picture of what is spoken of as "dumb chorea." There is a child in Alexandra Ward at the present time who for five weeks never spoke a word. She is now recovering, but the recovery, as is usual in these cases, is very slow indeed. Such patients are not unconscious; one recognises that they hear what is said to them and clearly are not comatose; they seem to be merely completely apathetic, and cannot be aroused by anything that is said to them. They usually continue to take food, and even in that condition they may sleep fairly well. Together with this mental condition there are usually obvious rheumatic phenomena of other kinds, often a progressive heart lesion, and the child to whom I have already referred, and who is now in Alexandra Ward, developed a series of rheumatic nodules which were felt along the tendons of the palmaris longus on both sides and on the elbows. Meanwhile there were progressive heart lesions, which diminished considerably when the severe stage of the disease had been passed. The movements, as a rule, become very slight in cases of this kind. The joint phenomena are not prominent. Of course one realises that in children the arthritic manifestation of rheumatism is a minor one, and I cannot recall a single case of chorea in which the pain in the joints caused acute suffering owing to the presence of the choreic movements. The heart troubles are much more prominent.

In the more active form of mental disturbance you may find every stage from excitement up to actual acute mania. Such phenomena, however, are much commoner in older patients, even up to fifteen or seventeen, and it is well never to look upon a case of chorea in a patient of such an age as trifling, however slight may be the symptoms at the onset. I remember very well enforcing this upon students many years ago, and admitting a boy of about seventeen years of age into hospital with very slight chorea, rather to emphasise what one was trying to point out than with any expectation that anything serious would result. And in that instance the lesson was justified by the course of events, for within forty-eight hours he required two male attendants to look after him and was maniacal. It is also well known that mental disturbance attending the chorea of pregnancy is often very acute indeed.

Another very important feature in some cases of chorea is the loss of muscular power. It happens at hospitals every year that a certain number of choreic patients are admitted on account of loss of power, either of the limbs on one side, or of the legs, or with general weakness. Such cases are sometimes diagnosed at first as cases of paraplegia or of hemiplegia. The movements may be very trifling indeed, and may be only noticed after the child arrives in the ward. The most obvious symptom is the loss of motor power, and that is a phenomenon which one must be on the look-out for, and which one must carefully distinguish from the apparent loss of power which is present in every case in motor chorea. The disadvantage under which the muscular movements are carried out is obvious if the child is told to grip the hand; the incoordinate movements put the child at a mechanical disadvantage, and so one notices an apparent weakness which is not really due to a loss of muscular power. The most obvious paralytic symptoms are seen in connection with the bad emotional or mental cases and during the period of convalescence. To refer once more to the case of the child which I have taken more or less as my text this afternoon, to-day after many weeks she begins to move her legs with some success. For a long time after the attack of chorea the child may be quite unable to make any active voluntary movement of the limbs, particularly of the legs. The general weakness is excessive, and there may be very great muscular wasting during the dumb or apathetic stage, perhaps partly due to difficulty in giving adequate food, but not wholly caused by that. Again, during the stage of convalescence, while the muscles are recovering, the general weakness may be very striking; the child has to be propped up in bed inch by inch, being quite unable to help itself in any way by the action of his muscles.

The condition of the knee-jerks in cases of chorea presents points of interest. There are four common types of knee-jerk met with in chorea. In some cases there is nothing abnormal to be observed, at any rate in the early stage. In a considerable number, also usually in the early stages, the knee-jerks are conspicuously increased.

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In others, again, the knee-jerks are lost. They may be lost in the middle of the attack or towards the end of it, or at any period. And their loss is not dependent on the administration of arsenic or any other drug. Children who are treated by rest in bed, without drugs at all, may lose their knee-jerks as suddenly and as completely as those who are on arsenical treatment. In the bad cases also, with the great muscular weakness and flaccidity, the knee-jerks are usually lost. The characteristic tendon reflex of chorea is what is known as the hung-up knee-jerk, in which the response is brisk and active but the relaxation of muscle is very much delayed, so that the limb is held lifted for a time and then falls only after a very noticeable interval. That is a much commoner phenomenon than is simple exaggeration, and commoner than simple loss. However, one cannot draw from our present knowledge any very definite conclusions, prognostic or diagnostic, as to the variety of chorea from the behaviour of the knee-jerks. Probably advancing knowledge will enable us to utilise such distinctions.

The time has now come to speak of the gravest accidents, not of chorea, but which tend to accompany chorea. These are the cardiac troubles Purely motor chorea is very seldom fatal. The cases belonging to the mental and emotional variety contribute some proportion of the fatal cases. The great bulk of the fatal cases are those belonging to the cardiac group. There are several ways in which the heart is apt to be affected in this condition. I have mentioned already the simple irregularity, which is called *chorea cordis*, and which is not of any gravity at all. Extremely interesting is that variety of cardiac affection, the recognition of which as a common phenomenon of chorea we owe to Dr. Lees and mainly to his work done in this hospital. That is the acute cardiac dilatation which is an early symptom in a very considerable proportion of cases in this malady. A child with simple choreic movements, not apparently ill, and without any murmur to be heard even on careful examination, may yet be shown, by careful percussion, to have a very conspicuous dilatation of the heart, as evidenced chiefly by enlargement of the cardiac area to the right of the sternum. Such dilatation is undoubtedly not a mechanical effect of the violent movements; it may be most conspicuous in cases in which the movements themselves are but trifling. It is a sign, as Dr. Lees and Dr. Poynton have shown, of myocarditis. They have taught us that myocarditis, instead of being a rare accident of acute rheumatism, is one of the common cardiac accidents associated with rheumatism, and that the physical sign of this myocarditis is a dilatation of the heart. As I have already said there may be no murmur, and in cases in which this dilatation is most pronounced there may be no murmur at all developed throughout the case. It does not necessarily follow that endocarditis is associated with such myocardial dilatation of the heart. The great importance of this particular phenomenon of chorea lies, to my mind, in the indication which it affords as to the desirability of admission of patients to hospital, or as to the treatment to be adopted in the cases of private patients. The presence of the dilatation is, I think, as a rule, the best indication that the child should be admitted and put to bed. I should attach far greater importance to it than I should to the violence of the movements, unless these are so violent that they are obviously beyond the proper control and management of the parents. A child who had very slight muscular movements but much cardiac dilatation I should treat with very much more care and circumspection than a child with very active movements and the heart within the normal limits. With the dilatation, or following it there may be the development of signs of endocarditis. The commonest of these is the systolic murmur, audible at the apex, conducted into the axilla, and due to a mitral lesion. In some few cases one of the earliest endocarditic signs is the development of what is known as the mid-diastolic mitral murmur, an apparent reduplication of the second sound limited to the apex. It is not at all an uncommon sign of early endocarditis, as well as being a common sign of mitral stenosis in a later stage. How these two statements are to be reconciled it is not very easy to see. It may be that there is an actual temporary stenosis produced by the thickening and swelling of the mitral valve curtains in connection with acute endocarditis. In addition to endocarditis and myocarditis there may occur pericarditis. Of all the manifestions of rheumatism pericarditis is the most malign. It is decidedly one of the less

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common manifestations, but at certain seasons it prevails with much greater frequency than at others, If you have one such case in a ward of a hospital such as this you are likely to have several. All the worst of the accidents of rheumatism are directly associated with pericarditis. Rheumatic pneumonia, as Dr. Cheadle pointed out, is so associated, and there can be no question that the rheumatic hyperpyrexia which played a very important part in the clinical picture of rheumatism forty or fifty years ago, but is now seldom seen, was intimately associated with pericarditis. Practically it never occurs without pericarditis. In the same way the occurrence of pericarditis in chorea is a decidedly malign sign, but it is perhaps one of the least common ones. Thus the myocardium, the endocardium and the pericardium, may be one after another attacked, and there may be a condition which my late colleague Dr. Sturges called "pan-carditis," in which all the structures of the heart became involved. You will remember from your experience and from what you have seen in the post-mortem room, or from what you have read in the literature, that in fatal cases of chorea the heart is hardly ever immune. I have never seen a post-mortem on a case of chorea in which there were no evidences of endocarditis, such as fresh beads formed along the edges of the mitral curtains, or some more extensive vegetations. That brings home to one the fact that these cases in which the heart is severely implicated are the worst cases of chorea, and those in which a fatal issue is most common. The worst cases of all are those in which the movements are great, the emotional disturbance extreme, and the cardiac troubles are also severe.

In conclusion a few words must be said on the subject of the treatment of chorea. I think we have been to a certain extent wrong in our aims in connection with the treatment of chorea. Our efforts have often been chiefly directed towards controlling the inco-ordinate movements, which, as I have endeavoured to impress upon you, are in no way the best indicators of the severity of the disease in the particular case. Some people have measured the results of treatment by the number of weeks the child spends in hospital, which I think is an extremely fallacious method of measuring or estimating results in any form of treatment. It largely depends on the degree of recovery which is considered necessary before a patient is sent out, the pressure on the beds, and various other causes. Many excellent observers of great experience have practically abandoned the active treatment of chorea at all.

In more recent years arsenic has enjoyed the chief reputation in the treatment of chorea. It is largely used in out-patient departments. There is no doubt that arsenic will cut short the movements of chorea, that is to say it will "scotch" and suppress them, provided you give it in toxic doses. Whether the end obtained justifies the means is, I think, a very open question. Personally I prefer not to give such large doses of arsenic to patients with chorea. The most reasonable course, it seems to me, is to treat these cases as you would other rheumatic cases, and to place our chief reliance upon the salicylates. Dr. Lees advocates the use of very large and repeated doses of salicylate, carefully guarded by equal or greater quantities of sodium bicarbonate, with the view to warding off any risk of the possible toxic effects which may proceed from such doses. And he has had very encouraging results in a considerable number of cases. Personally I am not in the habit of giving such large doses as Dr. Lees does, and though one cannot quote statistics giving definite results, I think it is a mistake to measure the action of drugs in different conditions by the same standard, and conclude that because a heart lesion does not tend to clear up and disappear in the way in which arthritis in rheumatism clears up and disappears, that therefore salicylates are of no value in warding off the cardiac complications of rheumatism. Believing fully that the symptom-complex chorea is a manifestation of rheumatism, I regard it as reasonable, in default of better treatment, to administer the salicylates in such cases.

In the bad mental or emotional cases other measures are often called for. Chloral is the drug which has enjoyed the greatest reputation in connection with such cases, and I have no doubt that in a good many instances it is very useful. But I do not think that chloral quite deserves the reputation it formerly had in this connection. I have seen some very remarkable effects in some cases from the use of sulphonal, and I believe one of my colleagues in this hospital has very great confidence in trional. Sulphonal is a drug which may produce severe toxic effects, even after it has

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been taken with impunity over long periods, and apart from any increase of the dose. Trional is certainly a much safer drug. Bromides are of little use in controlling choreic movements. They are probably useful in conjunction with chloral or alone, in controlling to some extent the mental disturbances which accompany the disease. In the cardiac cases special measures may be called for, such as ice-bags, blisters, etc. Whether the duration of the choreic movements is much influenced by drugs at all is, I think, an open question. If rest is insisted upon, choreic movements will practically always cease in time, but there are cases in which they persist for months, or even with relapses, for years. The late Dr. Andrew, who was one of my teachers at St. Bartholomew's, and one of the shrewdest physicians with whom I was ever brought into contact, used to say, in reply to students who asked him what was the best treatment for chorea, "Six weeks." And I think that in this he was right. I do not say that six weeks will see out all cases of chorea, but there are comparatively few cases in which rest for six weeks in bed will not bring about the necessary recovery. Recurrent attacks of chorea are, of course, common enough, and a child who has had it once is not unlikely to have it again at some future time. Moreover, a patient who has had chorea as a child may get a return of chorea years afterwards, if it be a woman perhaps during pregnancy, so that one attack of chorea by no means confers immunity from future attacks.

It has been my aim in what I have said to you this afternoon to try to impress upon you a wider view of chorea than sometimes prevails, to point out to you how many factors there are which go to make up the clinical picture of the condition, and to try to impress upon you that that upon which we are apt to concentrate our attention, the incoordinate movements, are not in reality by any means the most important features of the disease, nor those to the control of which our treatment should be chiefly directed. If I have succeeded in bringing this point of view home to you I shall feel I have fully carried out my intention in speaking to you this afternoon.

October 14th, 1907.

A LECTURE

ON

MEDITERRANEAN FEVER.

Delivered at St. Thomas's Hospital.

By F. M. SANDWITH, M.D., F.R.C.P.,
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GENTLEMEN.—To-day you will hear something about Mediterranean fever, which those responsible for the nomenclature of diseases have lately rechristened Malta fever. You may know it under both names, but neither of them are good, as I shall be able to show you when I speak of the geographical distribution of the disease. We must call it Malta fever to-day because the College of Physicians does so. There are two or three reasons for talking about Malta fever. One of them is, that if one is asked to lecture upon the tropical or exotic diseases of the world, I do not think it is of any use to choose those diseases which you have never seen and seldom hear of, and an example of which you are not likely to see. I think it is better to select a disease which you are likely to meet with, even those of you who are condemned by Providence to spend your lives in this country, and never get abroad to see what the real world is like. So one of the reasons I am talking about Malta fever is that it is a disease which may come to you, for there is a good deal of it in the world, and it is often undiagnosed. There is another reason why one should take more than an academic interest in it, and that is because I venture to hope it is one of the diseases which is going to disappear. It is certainly with us now, but I hope that in days to come people will be able to say to you: "This is a disease which you need not bother about, because we do not have it now." I shall tell you why it is that we have some reason for this sanguine view. You know you may talk of the intellectual development of man as, first, a period of ignorance or want of knowledge, secondly, a period of latent knowledge, and then a period of specialised and accurate knowledge. This disease, like most of those which you work at, has gone through these three stages. First there was ignorance about it, nobody knowing what it was.

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Later the micrococcus was discovered, and that may be described as the period of latent knowledge. People said that is the micrococcus which must get into the body, but how does it get out of one person's body and into another person? That could not be satisfactorily answered until lately, and now we may say we have special knowledge on the matter; we not only know that there is a micrococcus (and that was known some twenty years ago), but how it gets out of one human body and enters into another. That, you know, is a little further than we have got concerning the tubercle bacillus. Koch found the tubercle bacillus in 1882, and yet we, in 1907, do not quite know how tubercle is received; you can make a very shrewd guess, but you cannot be certain about it.

Let us look at the different names which are given to the disease. It is called Malta fever because it is connected with Malta so much. It was called Mediterranean because it was thought to be chiefly connected with the coasts of the Mediterranean sea. It was also called Gibraltar fever and Rock fever because Gibraltar is known as the Rock. Again, Neapolitan fever because it was found in Naples; it has been called Cretan, Cyprus, Danube, and Levant fever. The term "undulant fever" has also been given to it because the typical fever chart shows a series of undulations or waves. I show you some temperature charts which may remind you of two or three attacks of typhoid fever, with relapses, or of one attack of typhoid and one lengthy relapse and one short relapse. There are some names other than geographical ones, and I only name them to you as a warning not to use them. One of them is "gastric remittent fever," another is "bilious remittent," which is a term still used, but it does not mean anything. It has been called "intermittent typhoid," and that means nothing. Again, typho-malaria means nothing. Typhoid is one thing, malaria is another, but the combination name is a sort of illicit liaison which we do not recognize. "Fæco-malaria" may mean something, but I do not know what; "sewage fever" implies that sewage is the cause of it, which is not true either. So these terms are not to be perpetuated.

Definition.—It is an endemic specific fever, sometimes becoming epidemic, of long indefinite duration, with a tendency to long, wavy relapses; characterized by constipation, profuse sweats, anæmia, debility, neuralgia, and sometimes rheumatic sciatica and swelling of joints. The disease is dependent on a specific microbe, but is not directly contagious and is attended by a low mortality. Now as to the history. You know that in the history of ancient medicine everything seems to go back either to the Chinese or to Hippocrates. This time it is Hippocrates who wrote of long fevers occurring on the coast of Greece—and it is still very prevalent in Greece—with as many as four lengthy relapses, and, on one occasion, death on the 120th day. You can please yourself whether you consider that it was this fever or "relapsing fever," which I am not lecturing about to-day. It was not until 1859 that Malta fever was first described as a specific illness. In 1886 Colonel Bruce, R.A.M.C., found a special micrococcus and named it, after the island of Malta, *Micrococcus melitensis*. But, unfortunately, this disease is still somewhat confounded with malarial fever and with enteric fever. Enteric fever is also of long duration, but it is not very like Malta fever in its symptoms.

As to its distribution I have given you the names of some of the places which have named the disease. There are a few others, mostly seaports: Italy, Sicily, Sardinia, Constantinople, Tunis and Algiers. So far we have justified the name Mediterranean, but I shall show you that it spreads a long way from there. It is on the Red Sea coast, at such places as Suakin and Zanzibar, also the Punjab, Simla, Delhi, Agra, Allahabad, Assam, Hong Kong, Porto Rico, West Indies, and the Philippine Islands—a fairly big geographical range. It is only natural that I should not wish to exclude Egypt when I am touching on geography, because my experience has been chiefly there. This fever was unknown in Egypt until I found a case in November, 1883, in a young English lady who had travelled leisurely from England through Italy on her way to Cairo. The symptoms of her disease I found were similar to those which I had studied in cases at Gibraltar and Malta. Quinine in large doses had no effect on the temperature, which lasted a hundred days. Another proof, if you want one, that the diagnosis was correct was the fact that she did have enteric fever some years afterwards, in Cairo, when I again attended her. There have since been many sporadic cases in Egypt, occurring at Port Said, Suez, Alexandria, and also at Cairo, which, of course, is not a seaport.

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We found the Widal reaction (1 in 200) and by puncturing the spleen the *Micrococcus melitensis* was found and a culture was made of it. Clinically we saw that these cases were really those of Malta fever, because the febrile condition lasted two and a half months, and in two out of twelve cases at Port Said the micrococcus was found in the spleen. There have been cases also in the Sudan beyond Khartoum. One man contracted this fever on the White Nile, and his blood gave the positive result with the serum test. It has been found at Kassala, in the Sudan, amongst the Abyssinians. And lastly, there is one interesting case of this fever in a man who had never left England; this is the exception which proves the rule. He was a gunner, at Dover, who died on the 122nd day of his disease, and the serum test proved positive on the 68th day. The statistics of the British Navy for the three years ending 1903 gave 831 cases in the Mediterranean, of which 620 were attributed directly to Malta, 30 to Gibraltar, 30 to Corfu, 3 to Alexandria, and the remainder to other ports. So the British Navy suffered tremendously, and they got the disease chiefly at Malta. This is one of the things which troubled us with regard to the causation of this disease. We send our patients for a sea voyage, and we cannot conceive a healthier life than in a man-of-war, and yet the men, and especially the naval officers, suffered terribly from this disease. We could not tell how it was, and the fact that the officers got the disease much more in proportion than the men also perplexed us. They were better protected from mosquitoes and had better food than the men, and yet they were more attacked by the disease.

When we come to the causes of the disease it is due, as you know, to the entrance into the body of the specific micrococcus, and when you think of the *Micrococcus melitensis*, think for the rest of your life of another word beginning with "m," standing for milk, because we have only lately found that it is chiefly communicated by milk. There are three predisposing causes which are thought to be important, and even essential, in the matter: the sea coast, or a large river, exposure to sewage, and hot weather. You may notice that nearly all the parts of the world which I have told you of are places which are either on the sea coast, or are islands, or are on the banks of large rivers, and I have already told you that the disease is well known on ships in harbour, where, of course, there may be sewage all round them. There is no doubt it is a filth disease, but when improved sanitation occurred in Malta the disease was not eradicated. On the other hand, we were thrown off the scent for a time because in Gibraltar the well-known "rock fever" dimished and actually disappeared completely in the year 1904. This occurred shortly after certain changes in the sanitation of Gibraltar were carried out, and it was not unnatural, therefore, to assume that the diminution and disappearance of the disease were due entirely to the public health measures which were adopted. For instance, in Gibraltar the main sewer was lengthened, and greater attention was paid to the drainage of the houses, to street paving, and to scavenging. There was stricter supervision of food and milk and, most important of all, there was the formation of reservoirs in the rock for supplying all the inhabitants with pure filtered rain-water. They have rain at special times of the year, and they catch it in the large reservoirs in the rock for use in the dry season. But Major Horrocks, another Army medical officer, pointed out that twenty years ago the milk supply of Gibraltar came from the goats which had been imported from Malta; later, difficulties arose about importing these goats, and now goats are brought from Spain into Gibraltar, or else they are produced by local breeding on the Rock itself. The obvious lesson to be learnt from that is, that it was not the improved sanitation which was responsible for the change, but the alteration in the milk supply which caused the disease to disappear. Colonel Bruce has described the happy results of the Royal Commission which has been working since 1904 at Malta. This Royal Commission was appointed by the Royal Society at the request of the Admiralty and the War Office and the Colonial Office. This adds another feather to the cap of the scientific officers belonging to the Army and the Navy, because it is chiefly due to their work that the discovery which I shall tell you of has been made. It was known that the monkey was the only animal to take this disease naturally, except, of course, man. Still, some of the laboratory animals, such as the rabbit and the guinea-pig, could be forced to take the infection by various expedients, such as, for example, intra-cerebral inoculation. The Royal

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Commission examined various animals and among them the goat. The goat is very much in evidence in Malta, and supplies practically all the milk used. Flocks of goats in charge of a goat-herd are taken round there, and in the other places I have mentioned. They live in the streets by day and are shut up in pens at night, and are milked at the customers' doors. The customer thinks he is getting pure milk if he gets it in this way rather than if he buys it in tins at the shop and elsewhere. The Commission originally had very little hope that the examination of goats would yield any result. However, as a matter of routine several goats were inoculated with the *Micrococcus melitensis* and the result was watched. There was no rise of temperature and no other sign of ill-health. But in a week or two after inoculation the blood was found to be capable of agglutinating this specific micro-organism by the Widal test. Even then the goat seemed to be such an unlikely subject for the elucidation of Malta fever that the observation was scarcely continued; nothing more was done for several months, when by accident the matter came up again, and it was decided to repeat the inoculation experiments. A small herd of goats was procured for the purpose, and, as would be done by every scientific person, the blood was examined before any experiments were made upon them. To the surprise of the various members of the Commission the blood of these goats was found to react naturally to the agglutination test. This of course led to further examination and to the discovery that in the blood, in the urine, and in the milk of goats could be found the *Micrococcus melitensis*, that is to say, that the animal which was intended to be the medium of the experiments was found to be already suffering from, or had suffered from, this disease. Some thousands of goats in Malta were then examined and the great discovery was made that 50 per cent. of them responded to the agglutination test, and actually 10 per cent. of them were secreting the micrococci in their milk. Monkeys fed on milk from an infected goat even for one day invariably took the disease. I have told you that monkeys are susceptible. Now you have the further fact that monkeys got it by the alimentary canal, and they got it from the milk of goats which contained the *Micrococcus melitensis*. At this time there was an important experiment (an unintentional experiment) on the drinking of goat's milk by man. This was the case of a certain steamer called the "Joshua Nicholson," fully described in the reports of the Commission from which I am quoting. In 1905 this steamer shipped sixty-five goats at Malta to export them to America. I need not tell you that the captain and the crew rightly and properly drank the milk of the goats on the way over, and they drank it in large quantities, because sailors do not usually get milk on board ship. The result was that everyone who drank the milk was struck down by Malta fever. Sixty of the goats were landed—five of them having died on the voyage—and then they were examined, and thirty-two of them were found to give the reaction of agglutination, and the micrococcus itself was successfully isolated from the milk of several of these goats. So then at last there was discovered a mode of infection which explains many of the curious features in the epidemiology of Malta fever, the irregular seasonal prevalence, occurring at one time and one place and at another time and another place. It is most common in summer, very little occurring in the winter months, when there were no mosquitoes and very little dust, and therefore it was thought that the dust and the presence of mosquitoes might be a cause, just as we believe typhoid may get into the body by means of dust. But I do not propose to go through the various mental processes of the last ten years, because I want you to think especially of the milk question. It is true that in Malta and most of the other countries, which are all warm countries, there are more cases in summer, and this is partly explained by the temperature of the air being more favourable for the multiplication of the micrococcus, and also by the fact that in the summer there is more cream used with fruit and for ice-creams, etc. The fact of the existence of this poison in the milk explains now the curious fact that the officers and even the officers' wives suffered in a proportion which was about three times that of the men, that is to say, both soldiers and able-bodied seamen. The officer gets milk as a luxury in his tea and coffee, and the men do not. This fact also explains the isolated epidemics which have been from time to time reported. For instance, there was a case of a sergeants' mess, all the members of which were attacked by the disease, whereas the men living close by did not get it at all.

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That put out of court the possibility of the mosquito being the cause. The female mosquito does not care whom she bites, and when a group of people are attacked, and the remaining people outside are not attacked, there may be something different in their food supply which accounts for the attack. In this case it was the fact that the sergeants at their mess had goat's milk, whereas the others had no milk at all. When the milk discovery was made preventive measures in Malta were started as early as possible, in June, 1906. Of course the idea met with a certain amount of ridicule and prejudice and opposition, but by means of argument and persuasion the various regiments in Malta and the Naval and Military Hospitals agreed to banish goat's milk from their dietary altogether. Already there has been a very striking result. For six years, taking the last quarter of the year only, (because only that quarter is available for 1906), the average number of cases was 35·3 per 1000 of the strength. In the similar quarter of 1906, although that year began as badly as most other years, there were only 4·8 cases per 1000, that is to say, it was only about one seventh of the average which Malta had been accustomed to. There are other striking instances: for example, the Royal Naval Hospital at Malta had a very bad reputation, so bad that the men of the Navy when they were sick preferred not to enter the Hospital. When a man is very sick he is generally willing to go there to get well, but the Naval men found that if a man went in for bronchitis or with a broken leg he contracted Malta fever in the Hospital. But Malta fever has now been banished from that Hospital because the goat's milk has been excluded from it, and since that change it is extremely satisfactory to note that not a single case of Malta fever has been contracted in that institution. If you add together the total number of days which the men of the Army and Navy suffered in the Mediterranean stations you will find that in one year there were 70,000 to 80,000 days of illness due to this disease alone. If the absence of milk or the change of milk will succeed in stamping out this disease you will see at once that Malta, instead of being an unhealthy and dangerous station, will become a very healthy one, and we shall all agree that the Royal Society and the Members of the Commission may be warmly congratulated on the successful issue of their investigation.

There are two or three more points about the causation which I want to mention. The disease is never communicated to doctors or to nurses, so it is not strictly communicable from one person to another. The patient does not catch it from his next-door neighbour. Yet inoculation experiments have proved that men and animals can get this infection through the skin, and, therefore, it was suggested that the mosquitoes brought it, and that it was an insect-borne disease like malaria. But the milk as a factor in the production of the disease is so overwhelming in its evidence and importance that we can put aside the mosquito as having anything to do with the case. The disease occurs mostly between the ages of six and thirty, so that it is essentially a disease of early life. It seems to affect the sexes equally, but attacks strangers more than natives. Residence in an infected area does not confer immunity, though this can be gained for several years by an attack of the fever.

Now a few facts about the micrococcus: It is a very minute microbe. Cultures kept at a temperature of 22° C. still retain vitality after fourteen months. The micrococcus lives in milk for three weeks, in tap water for three days and even longer, but it dies immediately in sea water. In urine it can live from three to six days. Major Horrocks has found that it can live in dust for at least sixty-nine days, and on dried clothing for even longer, but exposure to the sun kills it in a few minutes. In that matter you are reminded of typhoid, especially in regard to urine, dust, and length of life. Experiments by feeding monkeys on the microbe will succeed, and one man is said to have been artificially inoculated by introducing it under the conjunctiva. The micrococcus has been isolated from the urine and from the blood, but never from the sputum, expired air, sweat, or from the fæces. You find the micrococcus in the urine and in the blood of infected people, and, as I have told you already, it is found in the milk of goats. In addition to infection by means of milk you must blame the urine, for it has been found in 10 per cent. of the urine of infected patients.

There are two types of this disease, with both of which you should be familiar. First of all there is an intermittent type, which may produce a temperature chart very similar to that of a person suffering from deep-seated pus or tuberculosis. If such a temperature were seen in a patient in a

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surgical ward an operation would be suggested. The other kind is the undulant, and I show you a chart of that.

Coming to the symptoms and course of the disease, you will find that obstinate constipation is a very important sign. Do not at once wonder why they find it difficult to diagnose it from typhoid fever; remember that constipation in this country and abroad is very often a symptom of typhoid. There are also extreme anæmia, debility, enlargement of spleen, and these are the only certain symptoms which accompany the fever, and those belong to typhoid too. But the indefinite duration of the temperature distinguishes it from all other fevers, because in severe cases the pyrexia may last six months, and convalescence may not be complete for two years. So the pyrexia is much longer than that of typhoid. The average stay of patients in hospital is seventy to ninety days. The fever is characterized, as I have told you, by intermittent waves; it is of a more or less intermittent type, a wave lasting one to three weeks, with an interval in which there is practically no fever for two or three days. The fever goes down enough to comfort both the patient and the doctor, and you can guess what the doctor feels like when the temperature goes up again and the friends begin to ask questions. The temperature in my cases has reached 40° C. or 104° F., but seldom higher than that. The highest record in these charts is 105·2° F. Many patients develop a tendency to enteritis, but the Peyer's patches and the mesenteric glands are never involved. When I was a student the examination questions often used to include: "How do you distinguish between typhoid and typhus?" Now we are emerging from the same condition of confusion of thought with regard to this disease as compared with typhoid. Profuse sweating is often present, and that accounts for the various names which have been given to the condition by the Italians, such as *febris sudoralis*. There is emaciation and loss of hair from the prolonged fever. The incubation period varies between two and ten days; it is not often you can get absolutely certain dates to rely upon. The complications and the sequelæ are more important than the symptoms of the actual disease. The most common are rheumatoid affections of joints, such as the hips, the knees, or the shoulders, sciatica, and respiratory diseases. You therefore have a rheumatic side in the sequelæ or a respiratory side. The respiratory diseases include bronchitis, pleurisy and pneumonia. When the respiratory system is affected and you have a long-continued hectic fever of the intermittent type, with sweats, you cannot help thinking of the possibility of tuberculosis, and tuberculosis has sometimes occurred after this disease. It is not likely to do so in a healthy person, in fact it cannot do so unless there is a double infection.

Now as to the morbid anatomy. I have seen a good many autopsies on cases in Malta and elsewhere, and one always finds the same sort of lesions. The spleen is enlarged, hyperæmic and soft, weighing 10 to 30 oz. The liver is generally somewhat enlarged and congested. The lungs usually show hypostatic congestion, and the kidneys are also hyperæmic. The small and large intestines are congested and this congestion is often seen to be in patches. There is no swelling or ulceration of solitary glands or of Peyer's patches, nor is there, in the ordinary run of cases, any enlargement of the mesenteric glands. So that often cases which have been called typhoid during life turn out post mortem to have been examples of Malta fever.

Diagnosis.—Mild cases with only a slight amount of fever are often overlooked. In a country where the disease is endemic the case is probably treated first as one of dyspepsia or as one of debility. In countries which are noted for malaria you detect that it is not malaria by the absence of the parasites from the blood and by noting that quinine does not affect the temperature. In order to distinguish cases from hectic fever of tuberculosis or liver abscess, you must rely upon absence of leucocytosis, bearing in mind that phthisis is sometimes a sequela of this disease. The effusion into the joints and the neuritis must be distinguished from acute rheumatism, synovitis and neuralgia. But the chief difficulty in regard to diagnosis occurs at the beginning of the case, when enteric fever is suspected to be present. You will diagnose Malta fever by the milder and longer course of the disease. In Malta fever there is none of the tremendous headache at night which is present in typhoid fever, nor is delirium present. Also there is no eruption in Malta fever, but there are profuse sweats, even during the fever. There is also the further additional help that the patient is in, or has come recently from, a place where the disease is

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fairly well known. Of course the serum diagnosis of late years has been a very great help. Those who have been brought up to be familiar with serum and diagnosis by its means can scarcely appreciate the difficulties which we had in the pre-serum days. When you have a doubtful case now you straight-away examine with two sera, the Widal test for typhoid and the Widal test for Mediterranean fever, and in that way you would diagnose the condition without trouble. When you have or hear of cases that give the double reaction, that is to say, the reaction to the test for typhoid and for Mediterranean fever, I advise that the culture should be changed, because if you keep these substances long enough they deteriorate. Therefore you must take particular care to have the sera quite fresh.

I now want to say a few words about prognosis. Although it is a disease which has such a long course and makes the patient feel so ill, it is not a fatal disease; only 2 per cent. of the patients die of it. And the majority of the deaths from the disease occur during the first six weeks of the attack. The average duration of the fever is about sixty days, but it may vary anywhere between fourteen days and three hundred days. The co-existence of diarrhœa, vomiting, and any other chronic exhausting disease, of course makes the prognosis dangerous. A sub-normal temperature lasting for a few days, and accompanied by a clean tongue and a return of the appetite of the patient, are the sure signs of an approaching convalescence. I saw a case not long ago, in Sir Patrick Manson's wards, who had been in Malta and caught the disease. There was no doubt about the diagnosis, and it was confirmed by the Widal test by different people. His fever continued, and he got worse and worse. However, I comforted myself with the reflection that if these cases do not die in the first six weeks they generally recover, whereas this man had gone on to several months. But he died, and there were no symptoms which I could discover during life except the continued fever with gradually increasing anæmia and debility. But when he was dead we found he had died of ulcerative endocarditis, not of Malta fever.

Then with regard to treatment. The patient must be, of course, removed from any insanitary surroundings, and he must, indeed, be treated in every way as if he were suffering from enteric fever until you are certain it is not enteric. You treat him as an enteric patient because this is the more important disease judged from the point of view of mortality. The patient must be confined to bed as long as the symptoms are persistent. The drug treatment is entirely symptomatic. The diet should be regulated according to the height of the fever and the condition of the tongue, but one may say that it is not necessary to restrict the diet for Malta fever to such an extent as is necessary with enteric. The rheumatic symptoms will require warm clothing and cotton-wool round the joint, and the patient should sleep between blankets. Open-air treatment will help the convalescence of the patient, and he should be sent to a warm climate for the winter months, though he should not be allowed to travel while the acute stages last. The only drug which is of use and worth mentioning is liquor hydrarg. perchlor., given in doses of one drachm three times a day. Possibly, however, it is only useful because it acts as an intestinal antiseptic.

Lastly, I must say a word about prevention. First, either exclude milk from the diet or be satisfied that the local supply is pure, or else boil the milk. Of course cow's milk can be imported if one is convinced that it comes from a clean source. In a place like Naples, where you do not know anything about the source of the milk, it is far better not to drink it at all. But if you feel milk should be used, take care to boil it, because that destroys the micrococcus. Other hygienic measures are also necessary, such as putting the patient in a sanitary house, and camps for troops should be carefully chosen. Avoid the bites of insects wherever the fever is prevalent. Also, as in the case of enteric fever, you should carefully disinfect the fæces and the urine, and every care must be taken with regard to the water-supply and general hygiene. As in typhoid, again, all attendants on the sick must carefully disinfect their hands. You know how important such disinfection is in typhoid fever, and it is equally so in Malta fever now we know that infection is conveyed by the alimentary canal chiefly, and especially by means of the milk which is drunk.

October 14th, 1907.

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ATROPHIC RHINITIS,

WITH SPECIAL REFERENCE TO THE ETIOLOGY AND TREATMENT OF UNILATERAL CASES.

By C. A. PARKER, F.R.C.S.Ed.,
Surgeon to the Throat Hospital, Golden Square.

ATROPHIC rhinitis may be defined as a disease due to a series of degenerative changes of the nasal mucous membrane, resulting in abnormal patency of the nasal fossæ and accompanied by a purulent or muco-purulent secretion, which tends to collect, dry into crusts and undergo putrefaction, and thus to produce the most distressing symptom of the disease, viz. ozæna, by which name the disease itself is often designated.

It is important to remember that the retention, drying and putrefaction of the discharge is chiefly due to the abnormal roominess of the nasal cavities, which renders "blowing the nose" ineffectual for the purposes of cleansing. The abnormal roominess is often greatly increased by anatomical peculiarities.

Atrophic rhinitis is usually bilateral, but unilateral cases are by no means infrequent. The object of this paper is to suggest that the unilateral form differs from the bilateral form both etiologically and clinically, and that it may be relieved by surgical treatment.

Etiology and clinical history of bilateral atrophic rhinitis.—Apart from a few cases due to destructive processes within the nose commencing in adult life, bilateral atrophic rhinitis is the outcome of chronic purulent rhinitis of childhood, which causes various degenerative changes in the nasal mucous membrane. Such changes include (1) the replacement of the columnar ciliated epithelium by squamous epithelium, (2) atrophy of the mucous glands, (3) round-celled infiltration with the formation of fibrous tissue, (4) thickening of the small arteries and obliteration of capillaries, and, probably as a result of these changes, (5) arrested development or actual atrophy of the inferior turbinals, causing undue roominess of the nasal cavities.

It is difficult to say at what point chronic purulent rhinitis ends and atrophic rhinitis begins, but the chief symptoms of the latter, namely crusts and stench, are usually first noticed at puberty, at which time there is a rapid increase in the size of the nasal cavities, adding to their roominess and rendering expulsion of the discharges impossible. In a few severe cases, however, these symptoms may occur very much earlier.

Etiology and clinical history of unilateral atrophic rhinitis.—In unilateral atrophic rhinitis there is seldom, if ever, a history of purulent rhinitis in childhood, and the symptoms do not commence until well after puberty—that is, not until some time after full development of the upper respiratory tract has taken place. There is always marked deviation of the septum, causing considerable and often complete nasal obstruction on the convex side and rendering the concave side abnormally roomy. The roomy side is naturally the one on which the symptoms of atrophic rhinitis occur. The inferior turbinals do not show the same amount of wasting or arrested development as they do in bilateral cases. Finally, the commencement of symptoms can often be dated from the occurrence of severe catarrhal rhinitis or from an attack of influenza.

The etiological factors of unilateral atrophic rhinitis are therefore probably as follows:

(1) Deviation of the septum causing nasal obstruction on the one side and excessive roominess on the other, which abnormalities do not reach their greatest proportions until after puberty.

(2) The establishment of chronic muco-purulent discharge as a result of acute rhinitis or influenza.

(3) Drying of the muco-pus into crusts, due in a great measure to the fact that, the opposite side being occluded by the deviation, the whole duty of warming and moistening the inspired air is thrown on the patent side, and hence an undue amount of moisture is abstracted and the discharges become dry and adherent to the mucous membrane.

(4) Roominess of the affected side preventing the expulsion of these dried discharges by blowing the nose.

(5) Putrefaction of the retained crusts leading to ozæna or stench.

In rare instances unilateral atrophic rhinitis may commence in childhood as a chronic purulent rhinitis, just in the same way as does the bilateral form. The deviation then saves the

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narrower side from the worst symptoms by rendering it possible to keep it clean by blowing the nose. In such cases the inferior turbinals will be quite small and seen with great difficulty, and muco-pus and possibly small crusts will be present in the narrowed nasal cavity. Unless, however, the combined width of the two fossæ be unusually great, the treatment about to be recommended for unilateral cases will be of service, as it will make it easier to keep both nostrils clean and free from crusts.

Treatment.—The treatment of ordinary atrophic rhinitis is unsatisfactory, for it is impossible to restore the inferior turbinals to their natural size or to render them physiologically active. Nevertheless, if the patient will persevere and put up with temporary discomfort much may be done to relieve all the symptoms, and especially to prevent the formation of crusts and to cure the stench.

All methods of treatment are based upon the necessity for keeping the nasal cavities absolutely clean, and for preventing the retention, drying, and putrefaction of the discharge. The simplest method, however, of attaining these ends is by first systematically cleansing the nose and then packing it with cyanide gauze. In the first instance the cleansing must be carried out by the surgeon. The crusts should be loosened by the application of peroxide of hydrogen and then the nasal cavities must be syringed until it is found by inspection and examination that every particle of crust and all discharge have been removed. When this has been satisfactorily accomplished the nasal cavities are packed with gauze, about eighteen inches of it being introduced on each side. The gauze should be removed at first night and morning and the nose syringed and re-packed. The patient will soon learn to do this for himself, and if it is done efficiently the crusts and stench will at once cease.

This treatment must be continued for many months, but eventually it will be possible to discontinue the packing and rely on washing alone. It is probable, however, that the patient will have to continue to syringe his nose at least once a day for the rest of his life, and this will be especially necessary after an acute nasal catarrh if he wishes to avoid a recurrence of unpleasant symptoms.

In unilateral atrophic rhinitis, in which the inferior turbinals are fairly well developed and more physiologically active the question arises whether it is possible to do anything to save the patient from the discomfort and inconvenience of packing the nose and of a prolonged course of syringing. The roomy nostril, the advent of a muco-purulent discharge, the inability to cleanse the nostril by blowing it, and the fact that the affected side is trying to do double duty in warming and moistening the air, are, as has been pointed out, the chief causes of the formation of crusts, their retention and putrefaction. If, therefore, the roomy side can be sufficiently reduced in size to make cleansing by blowing the nose possible, if the stenosed nostril can be enlarged and rendered physiologically active, and if, with appropriate treatment, the muco-purulent discharge can be relieved, then there should be fair prospects of, at all events, greatly relieving the symptoms without packing, if not of curing the condition. The first and second of these indications can be carried out by a Killian's submucous resection of the septum, and the third by the subsequent use of the appropriate local remedies.

I have now operated on a sufficient number of such cases to be able to say that this method of treatment at once cures the ozæna, prevents the formation of large crusts, and enables the patient to keep the nose clean by blowing, and that by the subsequent use of an alkaline nasal wash the discharge will greatly diminish in quantity and the patient can be kept free of all symptoms. Sufficient time has not yet elapsed to say whether, eventually, the nasal wash can be entirely dispensed with, but at any rate sniffing the lotion from the palm of the hand is quite sufficient to keep the nose clean and the syringe can be given up.

In performing Killian's submucous resection a little extra care is necessary in raising the muco-perichondrium from the affected side, as it does not separate quite so easily as in ordinary cases and tears a little more readily.

The following cases confirm the above statements and show the amount of success that may be expected :

Case 1.—E. H—, female, æt. 59 years. Symptoms, which were confined to the left nasal cavity, were first noticed at the age of forty-two, after influenza.

On examination the septum was found to be deviated to the right, causing considerable nasal stenosis. The left nasal cavity was very roomy

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and filled with decomposing crusts, producing the characteristic fœtor. The inferior turbinals were small but equal in size and quite visible. The patient had been treated in various ways, but without any permanent benefit.

A Killian's submucous resection was performed in the usual way.

Six months later the patient stated that she could cleanse the nostril by using her handkerchief, that there was very much less discharge and that there were no crusts and no fœtor. On examination a little muco-pus could be seen, but both nasal cavities were free from crusts and the mucous membrane was quite moist.

CASE 2.—R. M—, female æt. 32 years. Sym ptoms which were limited to the left side were first noticed at the age of twenty-eight. No history of nasal discharge in childhood. Very subject to acute rhinitis, but does not date symptoms from any particular attack.

On examination the septum was found to be deviated to the right causing considerable stenosis. The left nasal cavity was very roomy and filled with very offensive crusts. The inferior turbinals were small but easily seen.

Killian's submucous resection of the septum was performed.

Nine months later the patient stated that since the operation she had been much more comfortable, that the discharge had been much less and that there had been no crusts and no fœtor. On inspection a trace of muco-pus was seen in left nasal cavity, but no crusts.

CASE 3.—E. B—, female, æt. 29 years. Symptoms which were confined to the left side commenced at the age of nineteen. No history of nasal discharge in childhood. Had influenza about the time that symptoms commenced.

On examination the septum was found to be deviated to the right, causing almost complete obstruction. Left nasal cavity very roomy and filled with offensive crusts. No discharge or crusts in the right side. Both inferior turbinals well developed.

Killian's submucous resection was performed.

Six months later the patient stated that she had been very much more comfortable; the discharge had been much less and the fœtor had quite ceased. There had been an occasional crust, but of quite small size and easily expelled by blowing the nose. On examination the nasal cavities were quite clean.

CASE 4.—A. S—, female, æt. 22 years. Symptoms, which were confined to the right side, commenced at the age of seventeen, and apparently followed a bad cold in the head. No history of nasal discharge in childhood.

On examination septum found to be deviated to the left, causing nasal obstruction. Right side filled with offensive crusts, left side free from discharge and crusts. Both inferior turbinals easily seen and fairly well developed.

Killian's submucous resection performed.

Fifteen months later the patient stated that she had been much better since the operation. The discharge was much less, and though she got occasional crusts they were quite small and easily blown from the nose. There had been no fœtor. On inspection no discharge or crusts could be seen.

October 14th, 1907.

WE have received from the managers of the Oxford Medical Publications, Messrs. H. Froude and Hodder and Stoughton, the first two volumes of 'A System of Medicine by Eminent Authorities in Great Britain, the United States, and the Continent.' This work is edited by Professor W. Osler, of Oxford, and Dr. T. McCrae, of Baltimore. The publishers announce that this System will be completed in seven volumes, and, to judge from the list of contents in the circular issued, the whole field of medicine will be well covered by this work. It would be wearisome to enumerate the various subjects and their authors, but the scope and character of the work is well explained by Professor Osler in the opening article in Vol. I, on "The Evolution of Internal Medicine." He points out that this System of Medicine is designed primarily for the practitioner who wishes to keep himself informed of the existing state of our knowledge in clinical medicine. Elaborate discussions upon doubtful problems are avoided, and, as far as possible, a clear statement is given without unnecessary references to the literature of the subject. The paramount idea kept well in view all through is that the work shall be helpful, and, without doubt, that is the chief function of a System of Medicine. The hope of the profession is with the men who do its work in general practice, and the last word in the matter rests with the practitioner to make effective the new knowledge.

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CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE, AND CLINICAL REVIEW.

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A LECTURE

ON

SUBACUTE COMBINED DEGENERATION OF THE SPINAL CORD.

Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square.

By J. S. RISIEN RUSSELL, M.D., F.R.C.P.,
Physician to Out-patients at the Hospital, and Physician to University College Hospital.

LADIES AND GENTLEMEN,—The subject of this afternoon's lecture is subacute combined degeneration of the spinal cord, and I have an opportunity of bringing before you a patient with the malady in an early stage, so early that you will be able to recognise how readily a grave disease of the kind, which is probably never recovered from, may simulate a functional affection. A malady so grave as the disease which I have to speak of this afternoon may present so few signs that are definitely organic in the early stage that mistakes in diagnosis are very readily made, and the malady thus comes to be regarded as some functional affection of the nervous system, such as hysterical paraplegia or, it may be, neurasthenia. In addition to this there are various organic diseases of the nervous system which this affection closely resembles, so that it will also be necessary to discuss the differential diagnosis of these.

In order to facilitate matters I must ask you to allow me to begin by throwing on the screen a picture which will reveal the morbid anatomy of the disease, then we can dispense with the lantern and discuss the clinical aspects of the affection in the presence of the patient.

Subacute combined degeneration is a disease in which there are evidently two distinct processes going on in the spinal cord. We have evidence that there is a diffuse degeneration, which is evidently the primary morbid process, and that

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ascending and descending degenerations result in consequence of the destructive effects of the other morbid process. All parts of the cord are affected by the diffuse degeneration, but it specially selects the thoracic region, and in this region the white matter of the cord may be so much affected that nearly the whole of it may be involved by the diffuse degenerative change, so that only the grey matter with a narrow band of white matter immediately abutting on the grey are left intact. On examining sections from higher levels of the cord we find that the degenerative changes are less and less pronounced, and that while there is definite evidence of ascending degeneration in the dorsal tracts, such as are shown in this slide, there is less and less degeneration evident in the ventral region of the cord, though there is some to be seen in the direct or dorsal cerebellar tract, and in the ventral or crossed cerebellar tract of Sir William Gowers. In the ventral region of the cord some diffuse degeneration is seen in the region of the direct pyramidal tracts, but you may notice that it is not confined to these tracts; it is a diffuse degenerative change which oversteps the limits of the pyramidal tracts. What is true of the cervical region of the cord in regard to ascending degeneration being added to the diffuse degeneration is true also in the lower regions of the cord where descending degeneration is to be seen. Here is a slide from the thoracic region which shows that the degenerative change is very diffuse. The degeneration is diffuse in the ventral region and all through the dorsal tracts and lateral columns. On examining sections from lower regions of the cord, however, we find that in the lower lumbar and in the sacral regions there is practically no diffuse degeneration evident, the only degeneration seen being in the lateral columns on each side. Now and then you find a little degeneration in the region of the septo-marginal tract of the same character as the diffuse process which affects the cord more severely at higher levels.

There are one or two supplemental remarks that are necessary before we bring the patient in and study the disease from the clinical standpoint. In spite of the extensive degeneration which you have seen in the white matter of the cord, the grey matter escapes. Little or no change is evident in the grey matter. This is one feature of the disease that is striking, while another is that the degeneration is limited, more or less, to the spinal cord, so that when the medulla is reached less and less change is evident, and examination of the pons usually fails to determine any of the diffuse degenerative change that is so extensive in the spinal cord. All that can be found is degeneration in the course of the ascending tracts which are passing to the cerebellum, and which reach it by way of its superior peduncles. What is usually found is that in the medulla some diffuse degenerative change is seen, but that by the time the pons is reached all signs of diffuse degeneration have disappeared and there is only evidence of degeneration of the ascending tracts.

You have now seen enough to convince you that the disease is one in which gross changes occur in the spinal cord, that the changes are most pronounced in the dorsal region, but that other regions are involved to a lesser degree, and that two morbid processes are very definitely in evidence —a local destructive process, and ascending and descending degenerations which result from this.

Let us next consider the clinical aspects of the affection, and let me first give you the history of this patient's case. The woman is forty-nine years of age, and she has complained of loss of power in her legs more especially for one year, but she has felt tired generally and easily fatigued for two years, and the tendency to fatigue has been increasing during that period of time. During the same period she has noticed shortness of breath on exertion and palpitation. For the last six months walking has become so difficult that the patient has had to give up attempting to walk, even with assistance, and now she can only do so with great difficulty if assisted. In addition to this she has had, during the last six months, progressive numbness in her feet. Commencing in the feet, this numbness has extended so that it has reached the knees, and she has found that at times her fingers become dead, and are unduly pale and numb, even when the weather is not cold. This condition will come on when the hands are cold sometimes, but at other times it will occur spontaneously irrespective of any special exposure to cold. On rubbing the hands she finds that in half an hour or so she will get back life into the fingers, and with returning life she experiences a sensation of pins and needles in the various fingers which have felt dead previously.

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During the same period of time she has had a tight feeling in the abdomen at about the level of the umbilicus, but this has not been attended with any pain. She has had no pain in the trunk or in the limbs, and although there have been muscular twitchings in her legs they have not been accompanied by any actual pain.

Now as to her appearance: She tells us that she has always been pale, but that she has noticed that during the last six months the pallor has altered, that she has become more yellow than she was formerly.

There is nothing special that I have to call your attention to in her past history. There has been no antecedent disease that is specially noteworthy in her case. She has been a hard worker; she has had no miscarriages, and there has been nothing pointing to the possibility of syphilis. She has had five healthy children. Apart from a slight history of tubercle there is nothing noteworthy in her family history.

Now as to her present condition: In addition to what you can see for yourselves as regards her aspect, it is important that you should know that her cranial nerves are all normal. All the special senses are preserved, and the motor and sensory cranial nerves are all intact. There is not even any nystagmus, or nystagmoid jerkings of the eyes. I mention this specially for a reason that you will recognise presently.

As to the limbs, what is to be noticed is that there is a general enfeeblement, and that this enfeeblement is attended with loss of muscular sense, and that the sense of position is notably disturbed in the lower limbs. For instance, the patient's lower limbs can be passively crossed one over the other without her being aware that this has been done. They can be forcibly flexed at the knees, at the ankles or any other joints when she is blindfolded without her being aware of what is happening; so that there is very definite evidence of loss of the sense of position. When the patient is supported in order to allow her to walk, or rather to attempt to walk, her gait is seen to be spastic and ataxic. There is accordingly a condition of paraplegia, in which the individual, though able to make movements, does so in an incoordinate manner and reveals a spastic condition combined with the incoordination. Let us now inquire into the state of sensibility. I have already told you of what the subjective sensations are that she complains of. With regard to the objective defects, there is very little to be determined. She has slight blunting of cutaneous sensibility on the feet, and on the legs up to the knees. It is a little doubtful whether the defect extends a little higher up on to the thigh on one side, but you may take it that the legs below the knees, together with the feet, are the parts chiefly affected by a slight blunting of sensibility. Examination of her tendon jerks has revealed that her knee-jerks are exaggerated, but that she has no ankle clonus. The plantar reflex supplies us with the one definite sign in this case that we can say is beyond all question an indication of organic disease, but this sign has been so variable in this patient that I cannot tell you what you will see this afternoon, for you may see an extensor plantar reflex, or you may, on the other hand, see a flexor plantar reflex. It is because of this variation, and the further fact that this one little sign is so important in the diagnosis of organic disease, that I bring the patient before you this afternoon. I wish further to emphasise the fact that a grave malady like the one whose morbid anatomy you have seen thrown on the screen to-day may be present, when there is very little to permit of the diagnosis of an organic affection as opposed to functional disturbance of the nervous system. There is very little wrong with the patient's sphincters. She has slight difficulty in initiating the act of micturition, but there is otherwise no sphincter defect. Examination of her blood has not revealed any obvious increase in the leucocytes, but her red cells are markedly altered in shape and size. No nucleated red cells have been found. Let me now try to demonstrate one or two of the points to which reference has been made. You see that she can perform all of the movements of her legs while she is recumbent although there is the difficulty about locomotion. You also see that she has marked exaggeration of the knee-jerks, and that in spite of this there is not the slightest sign of ankle clonus on either side. While dealing with this part of her case I would ask you to notice that the muscles are wanting in tone, that they are flabby, and that there is also a general failure in her nutrition without any localised muscular atrophy. What is true of the legs is also true of her arms.

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Now, ladies and gentlemen, I want to show you how the plantar reflex is behaving, for, as I have already suggested, we may find that the response is flexor, or we may, on the other hand, find that it is extensor. You see that the response is extensor on both sides, for when the sole of the foot is stroked gently the great toe moves upwards towards the dorsum of the foot. This is an abnormal phenomenon as you are probably aware, and means that organic disease is affecting the pyramidal system in the spinal cord or elsewhere.

There is very little abnormal in the upper limbs for there is no notable ataxy, and the loss of power is only of a general kind such as may be met with in a case of neurasthenia. There is no definite paralysis or atrophy of any groups of muscles. As we have already seen that the cranial nerves are intact we are obviously dealing with an affection in which the lower limbs have suffered chiefly in consequence of the disease, and that the case is, accordingly, mainly one of paraplegia.

Let us next deal briefly with the ordinary clinical picture of the malady which you have seen here, and then with the differential diagnosis.

There is nothing very definite which we can tell you as regards the etiology of the affection. Some of these patients have had syphilis, others have been addicted to alcohol, in others some other toxic influences have been traced, and among them septic absorption, as there has been chronic suppuration in some cases. Then again, the disease has manifested itself in females after parturition, which has sometimes been normal, at other times not. In other patients there has been a history of rheumatism, while in others there has been a history of tubercle. Apart from these affections, which have sometimes been traced in other members of the family, there is nothing special as to the family history in these cases. Some belong to families in which there is tubercle, as in the case of this patient, in others there is a family history of carcinoma, and in others of rheumatism. Now a word as to the age at which the malady develops, and the sex that it is most prone to affect. The disease manifests itself usually about the middle period of life, as has been the case in this woman, and she is of the sex that it most commonly selects. There is not, however, any very striking difference in regard to sex, for men are almost as frequently affected as women, there being only a slight difference in favour of women—that is to say, they are a little more commonly affected than men.

Now as to the way in which the disease develops and progresses. In the earliest stages you find that, as in this patient, weakness is complained of. The weakness usually reveals itself by some slight spastic weakness in the lower limbs, in conjunction with which there may be ataxy. In some cases the ataxic element dominates the picture, in others it is the spastic element which does so. This, the earliest stage of the disease, is the longest of the three stages into which it is convenient to divide its course. The first stage is usually two thirds or three fourths of the length of the whole illness. In some cases it only occupies about half of the total duration, while the other two stages share the other half. In this stage, all that is present is a series of subjective feelings, such as numbness, tingling and so forth, with objective evidence of slight spastic weakness accompanied by ataxy.

The patient goes on in this way until, it may be somewhat abruptly, the clinical picture changes from this indefinite slight spastic paraplegia with ataxy to a very severe spastic paraplegia, in which spasticity is now pronounced, and by this time, if the patient has not previously had to take to bed in the way that this patient has, she becomes bedridden, for the spastic condition of the limbs prevents all possibility of standing and walking. Now it is that anæsthesia becomes developed, and beginning in the lower limbs it mounts up so as ultimately to reach the trunk. The spastic paralysis of the lower limbs thus becomes combined with anæsthesia, and may also be accompanied by twitchings of muscles, which may be associated with sharp, darting pains in the limbs. The patient at the same time may complain of a girdle sensation, if that has not been complained of previously, which sensation is experienced round the trunk at different levels and sometimes as low as the umbilicus. There may be nothing more, or while still in this stage the patient may complain of subjective sensations in the upper limbs, and there may be spastic weakness in those limbs with ataxy of the same kind as in the lower limbs. This spastic condition may be quite pronounced, as may be the ataxy, but more commonly the upper limbs do not show anything like the same degree of affection as is revealed in the lower limbs. As

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the case goes on the anæsthesia becomes more and more marked, the spasticity is accompanied by the usual exaggeration of the tendon-jerks, ankle clonus appears, and the plantar reflex is of the extensor type throughout.

Having lingered in this stage for a variable period, again somewhat abruptly, certainly in the course of a day or two, the patient passes from this condition of severe spastic paraplegia to one of flaccid paraplegia, with absolute abolition of all tone in the muscles. So that now, instead of exaggerated knee-jerks and ankle clonus, there is a condition of absolutely flaccid paraplegia, with knee-jerks and ankle-jerks both abolished. The exaggerated knee-jerks and the ankle clonus thus both disappear from the clinical picture. The plantar reflex, however, remains of the extensor type. Now it is that the patient suffers severely from anæsthesia, which becomes pronounced. There is profound blunting of sensibility—indeed, the anæsthesia may become absolute. In addition to this the sphincters now become severely paralysed, so that there is loss of control over the bladder and the rectum, and an evident tendency to trophic disturbance, which, in spite of the most careful and most skilful nursing, permits of the appearance of a severe bed-sore in the sacral region. This stage of the disease is rapidly fatal, for usually the patient succumbs in the course of a few weeks, five or six at most. There are exceptional cases, however, in which the individual will linger in this stage for some months. From day to day death is expected, but still the patient lingers on. There are some added features in the clinical picture to which your attention must now be called. In some patients there is a little disturbance in connection with some of the cranial nerves. Thus there may be nystagmoid jerkings of the eyes, slight in degree, on lateral movement. Sometimes the voice is altered a little, with some evident weakness of the vocal cords. A little tendency to weakness of one or other of the ocular muscles has also been noticed. As a rule, however, the cranial nerves escape as is the case in this patient, and they usually escape in this way not only early but even in the later stages of the disease. The only thing which is at all likely to appear is a little nystagmoid jerking of the eyes owing to slight weakness of the ocular muscles. Although the cranial nerves escape in this way, however, the brain becomes weak in the later stages of the malady. This is hardly to be wondered at, for by the time the brain symptoms appear the general state of the individual is exceedingly bad. The cerebral symptoms consist of a certain amount of mental wandering or delirium, which occurs especially towards the evening, but which may be perpetuated during the day as the patient becomes worse and worse, and as the final stage is approaching.

These cerebral symptoms are intimately associated with the very unsatisfactory general state of the patient. Rises of temperature are liable to occur from time to time. These elevations of temperature may occur even in the second stage, but if they have not occurred then they are almost sure to be met with in the final stage. When this stage is reached the rises of temperature may be accounted for by bed-sore, cystitis, pyelonephrosis, or other similar septic conditions which are liable to occur, but no such complication can be held responsible for the elevations of temperature which may occur in the second stage of the malady, and which do not then depend on any complications of the kind which are met with in the terminal stage of the disease.

The patients also become anæmic. You have seen this in the woman who has been before you this afternoon. Anæmia plays a very important and very prominent part in the clinical picture of the malady. There was a time when it was supposed to be the cause of the disease. It was thought that there was pernicious anæmia, and that the changes in the spinal cord resulted in consequence of the blood changes. Now, however, the more generally accepted view is that while some of the cases present the condition of blood which we ascribe to pernicious anæmia, in the majority of them the anæmia is of the secondary variety, and that it seems more likely that the anæmia and the cord changes are both due to a common cause rather than that the anæmia is responsible for the cord changes. In connection with this we have to consider the fact, that although anæmia precedes the cord changes in certain cases, there are others in which the cord changes antedate the anæmia, and that there are even some in which the anæmia is never a very pronounced feature unless it be at the very end of the illness. In this final stage of the disease, however, there is very definite marasmus,

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the patients become emaciated and cachectic, and look just like individuals who are suffering from the late stages of carcinoma. The condition of cachexia becomes pronounced. The patient may be severely anæmic, cachectic, emaciated, and may live on from day to day in a state of delirium, so that each day the end is expected, and usually comes, as I have already told you, in five or six weeks.

Now as to prognosis: There is only one case that we know of which appears to have recovered. The man was treated in this hospital. It is difficult to see what other diagnosis could have been made in the case, and yet the patient recovered and was able to return to work. Whether the diagnosis was at fault or whether the patient really suffered from the malady of which we are now speaking it is impossible to say, but no other case is known to have recovered. The usual course of the disease is such that death results in from two to three years after the onset of the symptoms. The course may, however, be much more rapid, and death has even resulted in three months. This woman has already had the disease two years, and at any rate for one year definite paraplegic manifestations have existed.

Let us deal with the differential diagnosis next. I cannot do better than first of all insist on the importance of the diagnosis of this class of case from a functional disorder. This very patient has not always been recognised as suffering from organic disease, for a competent observer, familiar with nervous diseases in general and with this one in particular, who happened to see the patient when the plantar reflex was normal and not of the extensor type which you have seen this afternoon, diagnosed functional paraplegia, and accordingly gave a favourable prognosis. You will thus recognise how easy it must be to overlook a case of this kind and to diagnose a functional affection when in reality an organic disease exists. The following case is also instructive in this connection:

A patient was admitted into another hospital under my care suffering from this disease. She had been sent to me by a former house physician of this hospital, a man well able to determine organic signs if any had existed when he examined the patient. He sent her into the hospital for a rest cure as she appeared to be suffering from neurasthenia, the result of overwork and worry. It was obvious that there was every reason to conclude that she was suffering from neurasthenia, and I could find absolutely nothing to cause me to form any other opinion until I reached the stage at which I examined the plantar reflex. On one side and on one side only the plantar reflex was of the extensor type, and the woman was dead in three months from the time she was admitted into the hospital. The one case is a good example of how hysteria may be diagnosed when in reality this malady exists, and the other shows how neurasthenia may be the only diagnosis possible in a disease that was to cause death in so short a time.

Apart from these functional conditions, of which nothing more need be said this afternoon, what organic affections of the nervous system may we confound with this particular malady? To begin with, if we are still to admit that there is such a disease as chronic myelitis—I do not refer to the condition which has been acute and in which the patient has been left with changes the result of the past acute attack, but to the affection which is described in the text-books as a chronic, progressive disease—I say that if we are still to recognise such a malady, then it must be very difficult indeed to find points to establish the diagnosis between chronic myelitis and the affection which you have seen this afternoon. I cannot pretend to draw a distinction from the clinical pictures of the two affections in so far as their spinal cord symptoms and signs are concerned. All that I can tell you is, that what used to be called chronic myelitis is now known to be explained by a variety of other conditions, and that the disease we are now considering is probably one of the affections which used to be called chronic myelitis before we knew anything about the morbid anatomy of sub-acute, combined degeneration of the cord. The clinical picture of this disease would do for what used to be described as chronic myelitis, so that I cannot give you any points of differential diagnosis between the two conditions.

We must next consider disseminated sclerosis. That is a disease which may be very easily confounded with sub-acute combined degeneration of the cord. Let us, therefore, see what points we must rely on to distinguish disseminated sclerosis from this other malady, because although they are both organic diseases remember that disseminated sclerosis is a malady which may permit a patient to

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live for many years, while subacute combined degeneration may result in death in a few months. The age of the patient is all-important. It is exceptional for disseminated sclerosis to begin at the period of life when this malady does. Disseminated sclerosis usually reveals itself earlier, and even when the more evident clinical picture is revealed after forty years of age it is often found on going back in the history that many years antecedent there have been indications of the existence of the malady, although the patient has not taken any special note of the symptoms. Then there is that emotional tendency which is so characteristic in disseminated sclerosis. You know how readily they laugh; sometimes they cry, but more usually there is a tendency for immoderate laughter to occur, which is quite out of keeping with the gravity of the patient's condition. There are also many symptoms which appear and disappear in disseminated sclerosis in a way that makes it seem that they must be hysterical in origin: a blindness that comes and goes, a diplopia which is recovered from, a loss of power in the arm or leg which exists for a time and then gets well. Many symptoms thus occur in disseminated sclerosis which come and go in a manner that suggests that they are functional, but this is not the case in this malady, which is usually steadily progressive. In disseminated sclerosis, apart from such symptoms as can be regarded as functional, definite organic signs of the malady may come and go in the most extraordinary way, so that the patient may at one stage of the disease reveal very definite signs of organic disease, and then, under treatment, the whole of these signs may disappear and the patient may recover completely, apparently. I have had an opportunity of showing in this very theatre a patient with disseminated sclerosis in whom the disease was so pronounced that there was inability to stand and walk, and yet that same patient subsequently was able to run up and down stairs. Organic signs may appear and disappear in disseminated sclerosis in a way which is not seen in other organic diseases. In the earliest stage of this disease a sign like that which I have just shown you may vary from day to day, and even at different times on the same day, but it does not happen that when such a sign becomes pronounced and definite it afterwards disappears and a normal state of things is re-established. The only changes that occur are such as I have just referred to, other examples of which are that the knee-jerks, which are at first exaggerated, may be abolished, and the ankle clonus may disappear. In the optic nerves we have a sign which is most important in diagnosis. The pallor of the optic nerves, which is so common in disseminated sclerosis, is not seen in the disease we are considering. Accordingly optic atrophy is a most important aid in the differential diagnosis. Furthermore, in disseminated sclerosis there is no cranial nerve that may not be paralysed, whereas it is exceptional to get any of them affected in cases of subacute combined degeneration. I have had to say that there may be a little nystagmoid jerking of the eyes in cases like that which we are discussing to-day, but there is never the pronounced nystagmus which may be met with in disseminated sclerosis, and therefore pronounced nystagmus should have some weight in the differential diagnosis, although this physicial sign is robbed of some of its value by the fact that a little nystagmus may sometimes be seen in cases of the other malady.

It will now be profitable to examine the clinical picture of subacute combined degeneration, to see what abnormal conditions occur that are not expected in disseminated sclerosis. Pains of a darting character in the lower limbs, which may occur as part of the clinical picture of subacute combined degeneration, are quite exceptional in disseminated sclerosis. A fixed, boring pain in the back and some dull, deep, aching pain in various regions may occur in the latter disease, but sharp lancinating pains which simulate those that occur in tabes are out of place in the clinical picture of insular sclerosis. In disseminated sclerosis anæsthesia is usually slight if present at all, and it is liable to be patchy and fleeting. Pronounced anæsthesia such as is met with in subacute combined degeneration is out of keeping with the most usual clinical picture of disseminated sclerosis. In the final stages of disseminated sclerosis gross and lasting anæsthesia may be determined, but even then it is not usually so widespread or so pronounced as in the other disease. The pyrexia which occurs in subacute combined degeneration without any evidence of septic absorption to account for it is not encountered in disseminated sclerosis. If a rise of temperature occurs in disseminated sclerosis there is some septic condition such as cystitis or pyelonephrosis

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to account for it. Anæmia has no part in the clinical picture of disseminated sclerosis, nor do patients suffering from this disease become cachectic, or waste to the same extent as may those who fall victims to the other malady. The profound anæmia, the cachexia, and the marked wasting that occurs in the later stages of subacute combined degeneration, are not special features in the clinical picture of disseminated sclerosis, indeed, they are usually foreign to it.

Turn your attention to tabes now, and first note what may lead to the belief that the patient is suffering from this malady. Tabes has often been diagnosed in the early stage of a case of subacute combined degeneration, when the patient has complained of numbness and tingling in the lower extremities, and when there has been slight ataxic weakness in the lower limbs without any spasticity. Tabes has also been diagnosed in the later stages when the knee-jerks have been abolished. A further similarity between the two diseases is supplied by the lightning pains and the girdle sensation, and also by the anæsthesia and analgesia. Besides which the muscles are in a hypotonic condition in both affections.

In spite of the similarity between the two diseases, there are points of difference that ought to prevent mistakes in diagnosis. In the earlier stages of the affections possibly there may be nothing to distinguish them except the plantar reflex. If the plantar reflex is of the extensor type the case is not likely to be one of tabes unless it is complicated, *e. g.* tabes combined with general paralysis, in which case there ought to be some other indications of general paralysis. If the case is one of uncomplicated tabes the plantar reflex ought not to be of the extensor type. In the later stages of the disease there is definite paralysis which does not belong to the ordinary picture of tabes. The patient affected by tabes may be unable to stand or walk, but this is due to inco-ordination, not to paralysis. When the patient is lying in bed the movements of the limbs can be performed with good muscular power, so that the detection of complete paralysis which is evident when the patient is lying in bed ought to prevent mistakes in diagnosis, in addition to which the past history will probably reveal that there has been spasticity at one time. In addition to this there are certain signs that occur in tabes which are not observed in the other malady. I refer to signs like the Argyll-Robertson pupil, which should be an aid, as it is present in a large proportion of cases of tabes. Many cases of tabes also have paralysis of the ocular muscles at some time or another in the course of the disease. Even at a late stage of the malady the extensor type of plantar reflex, which may prove helpful in diagnosis in the early stage, may be of value, for it ought not to occur in tabes, but is one of the most constant signs in subacute degeneration.

I now wish to refer to peripheral neuritis, as the difficulty in diagnosis may be even greater than in any of the other affections to which I have referred. Some patients suffering from subacute degeneration are seen at a time when the knee-jerks are abolished, at which stage of the malady the limbs are of course flaccid, and unless careful inquiry is made as to what preceded the flaccid condition it would be easy to fall into the error of diagnosing peripheral neuritis. On one occasion I nearly sent a patient into the wards from the out-patient room as a case of multiple peripheral neuritis, when it was really a case of subacute degeneration. The patient was sent to the hospital with the diagnosis of multiple neuritis, and with a history of alcoholic excess. She looked alcoholic, and presented flaccid weakness of the lower limbs with the knee-jerks abolished and her calves tender to pressure. I nearly confirmed the diagnosis that had been made, and would have done so had my attention not been arrested by the degree of anæmia present. Examination of the plantar reflex revealed the extensor type of response. That patient did not show any gross defect of sensibility at the time, but you should know that in the differential diagnosis between these two affections the degree to which sensibility is lost is a point of considerable importance. In addition to this attention should be paid to a history of spasticity before the limbs became flaccid, and careful search should be made for the extensor type of plantar reflex, all of which signs serve to distinguish subacute degeneration from peripheral neuritis. On testing the sensibility, a degree of blunting may be met with in this malady which is foreign to peripheral neuritis, especially in regard to the amount of blunting of sensibility which occurs on the trunk. The anæsthesia in peripheral neuritis is usually confined to the limbs, and

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is usually most evident in their distal portions. Finally, in the differential diagnosis we have the sphincter paralysis which, as I have already told you, comes on at the stage when you have this flaccid condition of the limbs. The sphincter paralysis which is usually so severe in the final stages of the disease further distinguishes the malady from peripheral neuritis, for the sphincters as a rule escape in peripheral neuritis. This is by no means an invariable rule, but even when the sphincters are affected in peripheral neuritis they are never so severely paralysed as in cases of subacute degeneration.

There are two other diseases to which I wish to call your attention before ending this lecture, as they may both be confounded with the malady we are considering. If enough attention is not paid to the history one of these cases might be easily mistaken for a case of acute myelitis. The time when the mistake is most likely to be made is when the patient develops spastic paralysis suddenly, after having had slight ataxy or slight spasticity previously. There is a liability to think that there has been a sudden lesion in the cord. In the same way, when the patient passes from the stage of spastic paraplegia to flaccid paraplegia the mistake has been made of supposing that the patient has been attacked by myelitis. A review of the history of the illness from its commencement will prevent mistakes of the kind.

I have left until last the question of the diagnosis of the affection from tumour of the spinal cord, for I have to admit to you that competent observers may fail to differentiate between the two conditions. This is unfortunate, for tumour of the cord may be dealt with successfully by the surgeon.

There is nothing more satisfactory in the surgery of the nervous system than when a tumour of the spinal cord can be diagnosed and localised, and when the indications are that it is extra-medullary in origin, for intervention can then be undertaken with every hope of success.

The clinical picture of the disease we are discussing may be very similar to that of a tumour of the cord, indeed, it may be so similar that, as I have told you, competent observers have made mistakes in the diagnosis. The points which deserve most attention in this connection are as follows: The severe root pains which are occasioned by a tumour do not occur in subacute degeneration. Darting pains, however, occur, so that you recognise that this is not a very strong point. Moreover there are cases of tumours of the cord in which the diagnosis has to be made in the absence of any notable root pains, owing to the fact that the tumour is not involving the posterior spinal roots. Nevertheless where there is severe root pain this must be allowed to weigh in favour of the diagnosis of tumour. Brown-Séquard's hemisection phenomenon is also of value in distinguishing these two classes of case. In this malady there is diffuse affection of the cord, so that the motor paralysis is manifested in equal degree in the two lower limbs, as is any evidence of sensory paralysis that is to be obtained. On the other hand, when a tumour is compressing the cord it is not uncommon to find that there is evidence that one half of the cord is compressed to a greater degree than the other, and when this is so the motor paralysis in one leg is in excess of the sensory paralysis in that leg, whereas in the other limb the motor paralysis is less than the sensory defect. This is known as Brown-Séquard's paralysis. A patient with a tumour pressing on the right side of the cord would have paralysis of the right lower limb, with slight blunting of sensibility on that side, whereas in the left lower limb the defect of sensibility would be more pronounced, while the loss of motor power would be less so. There is no other point on which special emphasis can be laid, so that there is not much to help us in differentiation of tumours of the cord from the class of case that has formed the subject of this lecture.

October 21st, 1907.

Our attention has been drawn to a discrepancy in our report of the Clinical Debate on "Gall-stones" at the Chelsea Clinical Society. Towards the end of the report Dr. Hale White is said to advise that in the great majority of gall-stone cases no operation should be done, whereas on page 279 Dr. Hale White sums up by saying "that olive oil and Carlsbad treatment should always be given a fair trial, and if they fail then the question should be seriously considered whether it would not be well to remove the gall-stones, not only to get rid of the pain of them, but because of the carcinoma and infective conditions that may follow."

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TWO LECTURES
ON
LEPROSY.

Delivered at St. Thomas's Hospital.

By F. M. SANDWITH, M.D., F.R.C.P.,
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LECTURE I.

GENTLEMEN,—You all know that leprosy is not strictly a tropical disease; it is more correct to say that it is an exotic disease. I need scarcely remind you that leprosy was once very common indeed in this country, that there are many records of leper asylums in the United Kingdom, but that is going back a long time. You will still find in old churches, for instance, at St. Mary's, Guildford, the little slits at the lower part of the wall facing the altar, being the places where the lepers were allowed to crouch outside and hear the service in old days. If you look at books on leprosy you will find any number of synonyms given, but I do not think you need trouble about them if you are going to remain in this country. But there are two or three names which I daresay you are familiar with, or with which you should be familiar, and one of them is the classical name lepra, which comes from the Greek *lepros*, meaning scaly or rough. Yet if you look at the pictures which I have brought you representing the disease you will see that the eruption is not a scaly one. So when the word *lepros* was used for the disease which we call leprosy there were doubtless many other skin affections included under the term. The Greeks called it by two names, both of which are sometimes used—elephantiasis and leontiasis. Elephantiasis Græcorum you will still find rather old-fashioned people talk about to distinguish it from the elephantiasis of the Arabs. True elephantiasis means the big swollen limbs or scrotum of which you have seen pictures. Leontiasis means that when the disease was advanced the man had a sort of leonine aspect, and his face was said to have somewhat resembled a lion's. I do not know whether you will allow that any of the patients shown in these pictures look anything like lions, but there are an enormous number of rugæ, marks and wrinkles, and an extreme number of nodules, rendering the face very unlike the ordinary human one. And those of you who have studied the nomenclature of the Greeks will know that they were sometimes very fanciful in the way in which they named different parts of the body. The French call the disease *lèpre* and the Germans *Aussatz*.

Definition.—We may say that it is a chronic human disease, of slow incubation, due to a specific bacillus, occasionally communicable to human beings in countries where it is endemic, characterised in one variety at first by thickening of the skin and then by the formation of nodules in the skin, mucous membranes and nerves, or, in the other variety by cutaneous erythematous macules, anæsthesia and invasion of nerve trunks. I am trying to give you a definition of the two ordinary varieties, the nodular form, sometimes called tubercular, and the macular anæsthetic variety or nervous type.

We must begin with the history. It has been stated, on the authority of Brugsch, a great Egyptologist, that leprosy was described in the Egyptian papyri so long ago as 2400 B.C. But the modern Egyptologists cannot agree that there is any real proof of the existence of this disease in Egypt in those very early days. So, although you may see it stated in books that the disease existed as early as that time, the diseases were then so conglomerate, so mixed up, that there is no definite proof that there existed in those days what we call leprosy. However, it is quite certain that there was leprosy in Egypt long before the Christian era, and that it spread to the Roman Empire—you will remember that Egypt then belonged to the Romans—in the first century before Christ. That is the first certain historic fact we have in reference to the disease. There are a great many mentions in the Pentateuch of leprosy, but there again it is probable that many other diseases were included, and it is even doubtful whether true leprosy was ever intended to be meant by those who wrote down those wonderful laws of sanitation. Those laws were probably recorded about 500 B.C., but although they were only then written down they were based on customs and tribal laws which were very much older. And if you re-read those chapters in Leviticus referring to leprosy you will find that other skin diseases were included amongst

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them, one being leucodermia, or whitening of the skin. In India, China, Persia and Greece this disease certainly existed before the Christian era, and it is still endemic in all those countries. Leper asylums and laws for the regulation of lepers have been known in Europe since the year A.D. 800, and the last point which I want to mention about the history, and not the least important, is that Hansen discovered the bacillus in 1871.

Now as to geographical distribution. In Europe there is still any amount of leprosy, chiefly in isolated centres, which are a legacy of the past history of the disease; there are little foci where the bacillus is still hanging jealously to the spots where it used to be so famous, for instance, in Spain, the Balkan Peninsula and in Iceland. In Great Britain there are usually perhaps 40 cases, but all of them are imported, generally from India, sometimes from Jamaica, for we do not grow our own leprosy nowadays, we have to get it from abroad. In France it is still met with on the south coast and in Brittany. In Holland there were 17 cases in 1897, and you must remember that Holland is still in close touch with the Dutch Colonies. In Belgium there have been 4 cases recently reported, in Denmark 3 cases. Norway was a home for it, but there has been a great decline in leprosy since compulsory isolation was established in 1885. In 1886 there were 2833 cases, whereas in 1896 they had dropped to 681, and it is believed by the Norwegian reformers that the disease in process of time will die out in Norway altogether. In Sweden 75 cases were reported, of which 30 were isolated. In Russia there have been many scattered cases, and there are still fourteen governments and nine provinces where leprosy is endemic and widespread. Many of these areas, but not all of them, are grouped round various seas or inland lakes, and that fact we shall look at again later when we come to causation, and the consideration of the fish theory. In 1897, in Russia, 1200 lepers were registered, besides many others which were not notified. In Finland at the same time there were sixty-seven cases. In Spain leprosy is attributed to the importation of infection from the Philippine Islands in 1850, but there are still the old asylums at Granada and at Seville; leprosy in Spain is chiefly on the south and on the east coasts. In Portugal there are about 500 cases. In Italy the disease is said to be increasing, and in one year there were seventeen deaths from leprosy. In Roumania there are about 200 cases, in Bosnia and Herzegovina 150, in Montenegro 100 cases. In Turkey it is quite common, but they do not trouble about statistics there; in Constantinople alone there are said to be 500 cases among the Jews. This is one of the diseases which affect Jews. You know that certain diseases seem to spare the Jews, but this is one which seems to hit them rather hard. In Greece there are a considerable number of cases, perhaps 200. With regard to Iceland there is a history of the disease having been endemic there since the thirteenth century, and even now there are about 200 cases. The fact that they have an increasing number of lepers has caused the legislature to pass laws for the isolation or segregation of the patients, the compulsory notification of the disease and the building of new asylums. Curiously enough, leprosy has been extinct in the Faröe Islands for 150 years, although the diet there is said to be exactly the same as among the Icelanders. There must be some local reason which we do not know which accounts for the disease being present in one place and not in the other. In the Mediterranean leprosy is still found in the Balearic Islands, in Corsica, Sardinia, Sicily, Elba, Malta, Cyprus and Crete. In Crete I have seen many cases; there are said to be 600 patients suffering from the disease there, mostly Greeks. Leprosy is also present in Mesopotamia, Syria, Asia Minor and in Persia since the early days, also in Arabia, Muscat, Siberia, Bokhara, and in China. In India it is chiefly in the belt of the Himalayas, in Kashmir and in the coast towns of India. But people also suffer from the disease away from the great rivers. In Ceylon there are some 300 or 400 cases. In the Malay Peninsula the disease is rife chiefly amongst the Chinese immigrants in Singapore and Penang. It is very frequent in Cochin-China, where segregation is employed. In the south part of Korea it is present, and it is very prevalent in Japan, one of the endemic centres, where there were 24,000 cases a few years ago. There were also many in Manila, but since it has been taken over by the Americans a leper hospital of 180 beds has been opened. In the Philippines there are some 4000 cases. In Australasia and New Guinea the disease is sporadic. In the Sandwich Islands leprosy was unknown

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until 1856, when it was introduced by the Chinese coolies; in 1882 there were 2000 people affected with the disease, or 5 per cent. of the native population. In New Caledonia it is present, and in Fiji, Australia (chiefly among the Chinese), and in New Zealand (chiefly among the Maoris). We now come to Africa. In Algeria there are many cases, which came from Spain. It is endemic in certain parts of Africa, such as Tunis, Morocco, Egypt, Abyssinia, British and German East Africa, Lagos and other parts of West Africa. In South Africa it came from the north before the white man settled there, and affected the Hottentots and Kaffirs. You have all heard of the huge leper asylum at Robben Island, which dates from 1845. Some of the photographs I show you to-day are from cases I saw there. It is a huge asylum, and one of the most interesting to see. In Basutoland the disease is present. There is an asylum also at Pretoria, and another at Emjanyama.

The total number of cases in South Africa as far as we can tell is about 2000. When it is possible to count the number of cases of a disease by thousands it is obviously time to legislate in some way to check its further spread. There are other places in Africa of less importance where the disease is prevalent, besides Madeira, Madagascar and the Seychelles. There are at least three foci of the disease in the United States—Louisiana, where leprosy has been known for more than a hundred years, Florida and Minnesota. In Minnesota there are perhaps 20 cases. It was suggested—but it is difficult to prove—that the people of Minnesota caught the disease from Norwegian leper immigrants. In Canada the disease is present in two or three counties, where the patients got it from the French settlers. In Mexico leprosy has been known since 1520. It is also present in British Honduras, Cuba, Jamaica, Trinidad, Barbadoes, South America, Venezuela, British Guiana, French Guiana, Dutch Guiana, and Columbia. In South America altogether it is said there are 27,000 lepers, but only 841 of them were segregated, in three asylums, in 1898. In Brazil there are over 5000 lepers. You may think that I have mentioned the whole surface of the globe, and if you look at this map you will see there are comparatively few countries where leprosy does not exist. But, as a whole, it is chiefly the southern parts, the warmer latitudes rather than the colder, in which it prevails. Probably we owe the absence of the disease in this country to the fact that there is something here which the leprosy bacillus does not like—perhaps it is too much cleanliness. Of course it is not the colder weather which keeps it at bay, because I have told you there are big centres in cold countries, such as Norway, Iceland, Sweden and Russia.

Now I want to say something about the causes. First of all we must mention the bacillus, and even if you have not yet a very great acquaintance with it you all know that there is a bacillus of leprosy, and you know that it very much resembles the bacillus of tubercle. It has the same reaction as that of tuberculosis, there are similar spaces between the rodlets. I show you photographs of the bacillus of leprosy magnified 1000 times, and of the bacillus *in situ* in a case of leprosy of the lungs, while another slide shows leprosy of the kidneys. The bacillus has the power of coming out of the leprous body; it comes away with the discharges, or with some of them, for instance from the sores of the leper. If you look at the skin surface of a leper who has only the anæsthetic form there is no sore by means of which the lepra bacillus can be brought away. So, in the same way, if you take a nodular case which has not broken down, the skin cannot allow the lepra bacillus to escape if it is fairly treated. But in the case of a patient with long-standing disease, with horrible sores, you can readily see that the bacillus can escape from the surroundings it is in. Many of these pictures show people who have definite sores. One patient has been photographed with her hands bandaged up, and probably the material with which they are bound is dirty. That is the sort of person from whom you would expect to get the leprosy bacillus. She looks like an old woman, but you must remember that leprosy makes all patients look aged; I should say a leprous patient looks about ten to twenty years older than his age. This woman looks sixty, but she was at the child-bearing age, because she had a boy and a girl born in the asylum. Remember that sores do not exist in the maculo-anæsthetic variety, but they exist in the nodules, and it is these broken-down lepromata which become sores. There is another very important part of the body where the bacillus of leprosy can be found, and from which it will escape into the outside world, and that is from the secre-

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tion of the nose. That was only found out a few years ago, in Germany, and since that has been known many cases have been examined and have proved to be leprous, although it was not quite certain, judging by the other clinical symptoms, that they were cases of leprosy. It is also important, as I shall have to tell you later on, to know whether the nose contains these bacilli, because it has been suggested by some that if a leper is not doing any harm to the community and is not capable of spreading the disease it is a shame to imprison him and put him upon an island, or in a hospital, or a retreat. One way of ascertaining whether a leper is dangerous to the community or not is to find whether the skin is intact, and secondly whether the patient has or has not the lepra bacillus in the secretions from his nose. Sometimes the bacillus is also present in the saliva, which of course may be associated with nasal secretion. We do not yet know whether the bacillus is dangerous to the respiratory and alimentary tracts of the healthy individual, but I think we would all take up the attitude that we prefer not to live with a leper and eat at the same table with him if we could avoid it. There is a secondary infection, perhaps through the blood or lymph stream, but certainly a secondary infection in some way. Then the question has been often raised whether we actually do know what the cause of leprosy is. If any of you said to me, "How can I become a leper without direct inoculation?" I could not tell you, although I could give you some shrewd ideas on the subject. Still, I could not guarantee that you would become leprous, even if you were to offer me a large sum of money should I succeed. And that is my personal test as to definite and certain knowledge of the causation of a disease. It is not sufficient to be able to cure a person who comes to you; you should in addition be able to say, "This is the way a healthy person can become infected," and the reverse of that is the way to prevent infection. As we do not know for certain the way in which the bacillus enters the human body, I can only tell you what we know about it, and that is largely a matter of theory. One suggestion which has been made of recent years is that the leprosy bacillus may enter by means of mosquito puncture, and you can guess that that idea rather came to the front after it had been found that mosquitoes were the agents or vehicles for the poison of malaria and yellow fever and one or two other conditions. It was suggested that possibly the leper became infected by means of mosquitoes, and it was pointed out that the primary lesion of leprosy—that is, the place where it is first discovered—is usually on some uncovered part of the body. That is more or less true, for it manifests itself early on the face, ears, hands and feet. When you look at these photographs you must not forget that some of these people who have leprosy on the body wear much less clothing than we do in a cold climate. But if you take any of these early cases you will see that the lepromata or nodules are chiefly on the face and arms and other exposed parts. We do not seriously think the mosquito does cause or carry the disease. You know, of course, that the mosquito naturally prefers to puncture on the face and other parts of the body which are exposed; they do not trouble to go through three thicknesses of clothing if they can get at exposed portions of the body. It is this which has led to the suggestion that the agent for carrying the condition is the mosquito. A French doctor, Dr. Noc, at the French convict station at New Caledonia, found that half the mosquitoes which had punctured lepers carried the bacillus of leprosy. That is an observation which, so far as I know, has not been confirmed, but it is interesting. It is quoted by Hallopeau. There is another suggestion besides that which I have just mentioned, namely, that lice may convey the bacillus, and it has also been thought that ants may. In Norway it has been pointed out that you not only have a combination of leprosy and mosquitoes, but that you have scabies. Leprosy, like many of the infections which you know about, is a dirt disease. The absolutely clean people of the world do not seem to suffer from it, but dirty people get it often. Now with regard to the possibility of puncture through the skin, there have been some interesting cases of accidental inoculation of the disease. People have got leprosy after accidental inoculation. There is, for instance, a case reported of a European boy in Borneo having developed leprosy nineteen years after a certain incident. He and a native boy were playing on the sand, and they vied with each other as to what each could do. The native, who was a leper, stuck a knife into his skin. His was anæsthetic leprosy, so probably that action did not hurt him. The European boy,

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not to be outdone, took up the knife at once and put it into his own skin. Nineteen years afterwards the European developed leprosy, and it appeared at that spot where the knife had been inserted. There is another case, that of a Sister of Charity who became a leper after pricking her finger while sewing a leper's clothes. People who are suffering from leprosy and have the thickened distorted hands which you see in these photographs cannot do any delicate operation such as sewing on buttons, and the Sister was mending the clothes and sewing on buttons when she stuck the needle into her finger and contracted leprosy through the puncture, and there have been many cases of leprosy which have been introduced through vaccination; the bacilli of leprosy have been found in the vaccine pustules in the old days of arm-to-arm vaccination. And there are other cases quoted by Dantec, a Frenchman, who has been writing on the subject recently.

Leaving now the possibility of the disease being carried by puncture through the skin, let us take up the question of personal contagion. It is idle to deny that leprosy is an infective disease, at any rate in a country where leprosy abounds. But apparently it is less infectious than tuberculosis. There are many instances of healthy people contracting leprosy in an infected country, that is to say in a country where leprosy is endemic, such as India, and then they communicate the leprosy to others on their return to a leprosy-free country. Thus you will find rare cases reported of people who have come back from India with the disease and who have been able to communicate it to their innocent friends who have never been outside this country. On the other hand there is considerable evidence that lepers may live in a household and never give the disease to any members of their family. You can imagine no closer intimacy than that which exists between man and wife, yet I have seen one woman who had had three leper husbands, and did not catch the disease from any one of them. I have here a note of an Egyptian who was a patient of mine, æt. 60 years, and he had had nodular leprosy for at least four years before I saw him. That is to say he confessed to a four years' history of the disease, and so it may have been there longer without his being aware of it. I questioned him about his marriage history, and found that he had had twelve wives, one at a time, and his present wife, whom I saw, was not in the least afraid of him, and none of his family, who knew that he had leprosy, contracted the disease. His wife, his sister, and a boy, æt. 9 years, all lived with him in one room, and I examined the skins of all and they were perfectly healthy. That is only one of many instances of a family living in close contact with a leper and not contracting the disease. One might narrate to you dozens of such cases. There are many things which appear to have nothing to do with the causation of leprosy, as far as we know, for instance, climate, altitude, and the nature of the soil. I have already mentioned that the disease exists in Iceland, and also in the Himalaya Mountains, so that cold alone is not enough to keep it in check. We must allow that leprosy shows a tendency to prevail along the coasts of seas, lakes and rivers, though in some parts of India, Russia, and Central Africa the disease is more rife inland than it is on the coasts.

There are certain predisposing factors, which are not, however, peculiar to this disease. For instance, dirt, insanitation, poverty, over-crowding and improper diet are always present. You will of course at once remind me that they are present also in the case of tuberculosis and many other diseases, or that they at least may be present. Mr. Jonathan Hutchinson, who has done more towards the elucidation of many problems than almost any other member of our profession, has lately revived an old theory about leprosy. I must tell you that Avicenna, who was a good old Arab, whose name has been distorted by Europeans from Ibn Sina, was one of the great men of the East as long as he was alive. He lived between the years 980 and 1010, and he believed that leprosy was due to fish, or was connected with fish in some way. And Jonathan Hutchinson has, of late years, strongly contended that fish, not fresh, but badly-salted and decomposing fish, is responsible for leprosy. Now it is quite true that many lepers have eaten this kind of fish or habitually do eat it, but in many parts of India there are lepers belonging to religions which do not permit them to eat fish. Hutchinson went to India, and he has also been to South Africa and other places, to work at this problem. And he says that when he came to individuals who belong to these non-fish-eating religions and who yet have leprosy, he always found that their religion had not prevented their

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eating fish. One does not understand why it is that people who live on the coast and can get fresh fish should eat preserved fish, but I must tell you that many people, such as Egyptians, who can get fresh fish, prefer the bad fish, perhaps like certain people who prefer to eat their game high. In Kordofan in the Sudan there are some lepers but there are no rivers, and therefore there are no fresh fish, and the inhabitants are so poor that they cannot afford to import preserved fish. At Gallabat there is leprosy, and the people believe themselves that leprosy is caused by eating fish. It is probably an old tradition which has lasted ever since the days of Avicenna. Still, in spite of all the respect which we naturally feel for Mr. Hutchinson, most of us say that until the bacillus of leprosy is found in fish, either fresh or preserved, we must consider that his theory is not proven.

No race is exempt from leprosy. It is not a racial disease, and in China, India and South Africa, though the natives suffer most from it, there are occasional cases amongst whites. I saw, for instance, Dutch lepers on Robben Island, as white as Englishmen, and there are people of British parentage who have suffered from it lately in South Africa.

With regard to age, the youngest case I have seen was that of a boy, æt. 5 years, who was the son of a leper, and the oldest was æt. 80 years. The average age of patients is a little over twenty.

Then we must look at the question of heredity. Leprosy cannot be transmitted like syphilis, and I think no one has ever seen a newly-born leper in the same way as one can see a newly-born syphilitic. In Egypt I saw a man, æt. 65 years, his son, æt. 35 years, and a grandson, æt. 10 years, and they all had leprosy, and all were living in one house. It was the nodular form in all of them, and I thought it was an interesting fact bearing on personal contagion; but that man had three daughters, and when he allowed me to see them I found that they were quite healthy. So apparently it was an accident that three members of the family, the males, had the disease, while the three females were spared.

October 21st, 1907.

SYSTEM OF MEDICINE. Edited by Professor ALLBUTT and Dr. ROLLESTON. London: Macmillan and Co.

The first three volumes of the new edition of the 'System of Medicine,' by Professor Allbutt and Dr. Rolleston, have now been issued by Macmillan and Co. The work fully maintains the high level of excellence attained in the first edition, and the editors must be awarded a large measure of praise for their labours. If any volume deserves more particular notice than the others it is the third, comprising general diseases of obscure origin and diseases of the alimentary canal. A fair idea of the practical value of this system can be drawn from a consideration of the following examples of modern teaching taken from these volumes.

Mr. C. B. Lockwood, in discussing the expectant and palliative treatment of appendicitis, observes that the cure of appendicitis by rest, dietary, and drugs is uncertain. This is obvious in the light of some of the causes of appendicitis: concretions, foreign bodies, bends, torsion, stricture, empyema, mucocele, tuberculosis, cancer, or actinomycosis. But it is reasonable to suppose that under favourable conditions some of the other causes of appendicitis may cease to act. For instance, fæcal or septic contents may be expelled into the cæcum; or bends or kinks may straighten out and allow the appendix to clear itself; or it is just conceivable that a torsion might become untwisted. Inflammation, too, of the mucosa, and especially of its lymphoid tissue and lymphatics, might reasonably be expected to subside, as it does in other parts of the body. The cure of ulceration is less likely, and the cicatrisation of ulcers may end in permanent stricture or stenosis. But no one can accurately infer the pathological conditions underlying an attack of appendicitis. In about 80 per cent. the cause is ulceration of the mucosa with septic contents and bacterial invasion. Therefore in every case the greatest vigilance must be exercised, and the prognosis given with diffidence. It is to be remembered that chronic appendicitis may, through want of care, become acute; and acute appendicitis may end in abscess or in septic peritonitis. At the end of the article Mr. Lockwood very truly says that it is to be remembered that surgeons are not infrequently called upon to remove the appendix as a prophylactic measure after the departure of acute attacks of inflammation. In such circumstances the microscopical sections may be normal, or show nothing but the faintest trace of the antecedent inflammation; but nevertheless it is unreasonable to reproach the surgeon for

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having removed a normal appendix. Furthermore, surgeons are not infrequently called upon to operate during the quiet stage, and when they are dependent upon others for information as to what occurred during the acute attack and for a correct diagnosis. But nevertheless it remains true that appendicectomy is one of the safest and most successful of abdominal operations.

Mr. H. L. Barnard, in a valuable contribution on "Intestinal Obstruction," states, in regard to the treatment of intussusception in babies, that the best results are obtained by rapid laparotomy and reduction when a surgeon is immediately available and the surroundings are favourable. Failing this, the best treatment is injection of warm water or milk by a rectal tube and funnel, with a drop of not more than 3 ft. The escape of fluid around the tube may be prevented by cotton-wool and vaseline and by holding the buttocks together. The child should be anæsthetised and the buttocks well raised. The tumour should be manipulated to assist reduction. It may be necessary to repeat the process twice, or even thrice, before it is successful. The child should then be placed under morphine and atropine to prevent recurrence, which is not uncommon; thus, of twenty-three cases treated by injection, fourteen required subsequent laparotomy. In adults, intussusception is seldom so urgent as in babies, and there is usually time to call in a surgeon, as the treatment is entirely surgical.

The teaching of Dr. C. J. Martin and Major G. Lamb on "Snake-poison and Snake-bite," in Part II of Volume II, should be widely disseminated. They observe that the symptoms following the bite of the European viper resemble those of a small dose of rattle-snake-venom. The bite is immediately followed by local pain of a burning character; the limb soon swells and becomes discoloured, and within from one to three hours great prostration, accompanied by vomiting and often diarrhœa, sets in. Cold, clammy perspiration is usual. The pulse becomes extremely feeble, and slight dyspnœa and restlessness may be seen. In severe cases, which occur mostly in children, the pulse may become imperceptible and the extremities cold; the patient may pass into coma. In from twelve to twenty-four hours these severe constitutional symptoms usually pass off, but in the meantime the swelling and discoloration have spread enormously. The limb becomes phlegmonous and occasionally suppurates. Within a few days recovery usually occurs somewhat suddenly, but death may occur from the severe depression, or from the secondary effects of suppuration. When a person has been bitten by a poisonous snake the efforts to influence the result should take two directions—to prevent the absorption of the poison, to counteract or lessen its effects on the organism. With the first object a ligature should immediately be placed on the limb above the situation of the bite; the ligature must, of course, be applied where there is only one bone, and not on the forearm or lower leg. Wall strongly recommends a stout indiarubber band as being by far the best material for use as a ligature; but in ordinary circumstances only a portion of clothing would be available, and answers quite well. The ligature must be tightly applied; this is easily attained by passing a stick under the ligature and twisting it. The action of the ligature is to delay the absorption of the venom for just so long as it may be applied, and in the case of venoms, such as those of the cobra and the krait, which contain no fibrin-ferment, this is all that is accomplished by its use. Having by means of the ligature insured that no more poison is absorbed, the next step is to destroy or remove what poison still remains deposited locally, with the object of preventing as much as possible of the venom from entering into the general circulation on removal of the ligature. Wall strongly recommends careful and deep dissection with the knife of all the parts likely to contain the poison. The dissection must be free in all directions, especially so in the direction of the lymphatic and venous return. In the case of the fingers, hand, and such parts, it should be carried right down to the bone. After this free and careful dissection the wound should be freely washed out with a strong solution of permanganate of potash, which will destroy any poison with which it comes in contact. As well as attempting to remove and destroy as much as possible of the venom which has been deposited locally we must endeavour to render innocuous any poison which has been already, or is likely to be, absorbed into the general circulation. Various remedies have been suggested and strongly recommended for the treatment of cases of snake-bite. Alcohol, ammonia, and strychnine have been strongly advocated, the two latter especially in Australia. But all experimental work, as well as practical experience, prove that none of these medicines is of the slightest use as an antidote to snake-venom. While all these empirical methods have failed us in the treatment of cases of snake-bite, recent research has shown that along the line of serum-therapeutics lies the true antidote for snake-venom intoxication. As regards the treatment of snake-bite other than by anti-venomous serums little or nothing can be done beyond keeping the patient quiet and warm. Small doses of alcohol may no doubt be beneficial as a stimulant, but this end can be more effectually gained by ammonia or strychnine, and the employment of enormous doses of alcohol cannot receive too strong a condemnation. In the present state of serum-therapeutics against snake-venoms more reliance should be placed on local treatment than upon the injection of any anti-venomous serum.

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GRAVES' DISEASE.*

By HECTOR MACKENZIE, M.D., F.R.C.P.,
Physician to St. Thomas's Hospital and to the Hospital for Consumption and Diseases of the Chest, Brompton.

LADIES AND GENTLEMEN,—Some two years ago I had the honour of giving a lecture at this college on the subject of Graves' disease, and showed by means of the lantern photographs illustrating its pathology and clinical features. I had not then time to discuss the course and the treatment of the disease as fully as I could have wished. So I thought it would be interesting to-day to show you some of the patients who are at present under my care and incidentally to say something about the different types met with and the course and treatment of this disease.

Graves' disease is one of those maladies for which physicians are constantly trying to discover a really efficient remedy. Although every now and then we hear that a specific has been discovered, as yet, I am sorry to say, we have found no real specific for the disease. We have many remedies, but no cure. Still, I have no hesitation in saying that by our treatment we can greatly help the patient in combating the disease.

The first patient I show you is a young woman, æt. 24 years, who is employed as a clerk in the City. She was sent up to me two or three weeks ago by her doctor because she has recently been suffering from palpitation and her eyes have lately been noticed to be becoming prominent. One found, however, that she had noticed a swelling of the neck for about a year, thus a considerable time before the heart began to trouble her. You will observe that as she unfastens her dress her hands are tremulous and that tremulousness has been present about six months, dating from an

* A Medical Consultation at the Medical Graduates' College and Polyclinic.

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illness which was thought to be gastric ulcer. This is a case which we may appropriately call slight as we see the patient at the present time. She has all the symptoms of the disease but in moderate degree. Each time I have seen her her pulse-rate has been about 120. There is a fine but obvious tremor of the hands. You can now see that the goitre is moderate in size, so that it might easily have been overlooked, and the patient tells us that it has not been larger than it is now. The eyes are not strikingly prominent, but they have a staring aspect. There is a slight retraction of the upper eyelid, and von Graefe's sign is either very slight or absent. The patient says she has lost about three stone in weight but is gaining flesh again now. Her skin is now moist and she tells us perspiration is very readily induced. She is continuing with her work at present and as long as she feels equal to it I should encourage her to do it. The fact that she is now gaining weight is a favourable point and I think she is likely to do well.

The next patient I show you was under my care in St. Thomas's Hospital from November 22nd, 1905, until May 4th, 1906. She has come up to see me at the hospital at intervals since then. She now looks fairly well and seems to be well nourished. Her hands are tremulous but only slightly so. Since she came into the hospital a year ago she has improved very much. She has never followed any occupation. Her age is 22 years. The history is that apart from anæmia when about eighteen years of age, for which she had treatment, she has had no previous illness. About the beginning of 1905 her mother noticed that she had a staring expression with some prominence of the eyes. On this account she went to her doctor whom she attended for three months. But she said she became no better and the prominence of the eyes got slightly worse. About the middle of 1905 she noticed that the collars she wore were becoming rather tight, and then it was observed by the doctor that she had a slight swelling in the front of her neck. The swelling she thinks has slightly increased in size since then. Lately the patient has felt unable to do her work on account of exhaustion and weakness, and has complained of her legs giving way. She has been rather short of breath, but has not noticed that she has been more nervous since her illness. For the last two or three months she has had occasional trembling of the hands, more especially after exertion. She has had some palpitation but not usually very bad. Lately she has had two severe attacks while asleep which woke her up, and these were accompanied by shaking so severe that she says the bed shook. Her hair has been falling out since Christmas, 1904. Occasionally she has been very low-spirited and hysterical. Lately her eyes have ached very much and the sight has become blurred after reading or other eye strain. At one time her face was rather puffy, especially about the eyes on getting up in the morning, but this puffiness disappeared after she had been up a little while. There has been no swelling of the legs. No history of shock or fright. When in the hospital it was noted that her nutrition was good. She seemed to be of a nervous temperament. The eyes were prominent. Von Graefe's sign was not present at the time and it is not now. The left palpebral fissure was larger than the right one, but now there is no difference. Blinking movements in her are not so readily produced as in a normal person, and as you see there is very little response after waving the hand in front of the eyes. The thyroid gland was then only slightly enlarged, and you will notice now there is very little evidence of enlargement. No pulsation was seen or felt over it. No thrill could be felt or heard. The skin was generally moist. There was a fine tremor of the fingers. The pulse was regular, 100 per minute. There was nothing abnormal about the heart. The first sound at the apex was rather thumping in character, the pulmonary second sound not accentuated. During the time she was under treatment at St. Thomas's she spent most of her time out of doors on the balcony. We also gave her a powder called rodagen about which I may say a few words. This substance, rodagen, with which I have been treating most of the patients whom I am going to show you to-day, is prepared from the milk of thyroidectomised goats by a German firm in Charlottenburg. A pamphlet issued by the manufacturers claims for it that it is a specific for Graves' disease, which claim I am quite unable to confirm. They describe it as containing 50 per cent. of "the active constituent" of the milk of these goats isolated by a process elaborated by Dr. Burghart and Dr. Blumenthal, in the Clinic of Professor Von Leyden, of Berlin. There is 50 per

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cent. of sugar of milk added to preserve it. I have tried it in a good many cases and in doses of from a drachm to three drachms a day. In some of the cases described in this pamphlet it has been given in even larger doses, and for considerable periods, but seeing that the substance costs wholesale between three and four shillings an ounce it is a matter of considerable expense to use it on a large scale. I have obtained from Mr. Jennings, the pharmaceutist of St Thomas's, the figures as to the amount we have used during the last two years and the cost. The amount is over 13,000 grm., or about 450 oz., and the cost has been £76. It is more difficult to tell you what amount has been used by my private patients or the cost, but the amount would probably be as much and no doubt the cost has been more. So that I think I may reasonably say I have given a fair trial to the substance, and still you will see that none of the patients who have had it can be said to be cured. Some of them are certainly better, but I cannot say that the results are very striking. I have a patient at present in St. Thomas's who has been confined to bed for a good while. She was a very obstinate case and has had the disease in a severe form. She did not improve for a long time and it was only after we had put her on rodagen that she began to gain weight. She has now gained a stone in weight since we began treating her with this substance. That is one of the cases which rodagen seems to have benefited, but the patient is not yet sufficiently well to admit of my bringing her here for your inspection. I may say that in no hospital case have I seen any bad effects from it, and that is certainly a point in its favour. One of my private patients who was taking two drachms a day thinks that it has caused diarrhœa. The lady said that after taking the powder her bowels became loose. She stopped it for a week and she had no diarrhœa, then she took it again and the diarrhœa recommenced. That is the only case in which any such effect has been observed, and I am doubtful whether in this case it was not merely a coincidence, seeing that attacks of looseness of the bowels are not uncommon occurrences in cases of Graves' disease.

As far as my observations go it seems to be a safe remedy which may be given in fairly large doses without upsetting the patient. Most of the patients say that they feel better, sleep more soundly and are less nervous while they are taking it, and some of them have gained weight after it. Still, I cannot say that the cardinal features of the disease, the exophthalmos, the goitre, or the tachycardia have been influenced to any very appreciable extent. This particular patient at the commencement of treatment was fairly well nourished. Under treatment she gained a stone, which she lost again when she returned to her ordinary mode of life. Her general condition improved as her weight went up and she felt better. With regard to the effect on the heart, her pulse at first was recorded as 100, 96, 84, 104, 88, varying between 84 and 100. After she had been under treatment for some considerable time with rodagen her pulse still varied between 100 and 94, so there was no striking difference in that way. At the present time she still has a rapid pulse. I have just counted it and it is 112. This patient, then, in spite of fairly prolonged treatment with the substance, has remained practically *in statu quo*.

Another substance which has been used and is still advertised is what is called "antithyroidin-Mœbius," prepared by Merck, of Darmstadt; it is described as the blood serum of thyroidectomised rams, the thyroid glands having been removed six weeks previous to the first bleeding. It is an even more expensive remedy than rodagen. I think 10 c.c. of it costs about five shillings, and the average daily dose is 5 c.c. It is given by the mouth, but may be given hypodermically or *per rectum*. I made a very fair trial of it, and I came to the conclusion that in the doses recommended it had no effect one way or the other. Here is a little book descriptive of Merck's specialities, and you will find "antithyroidin" referred to as a tested remedy for the treatment of Graves' disease. Tested but found wanting is the way I should put it. Here is a little pamphlet which is issued by the same firm describing the good effects of this remedy. One German physician says "that from an experience gained in a very interesting case he is of opinion that the remedy taken in slowly increased doses exercises fairly rapidly an important and often an unexpectedly favourable influence on all the symptoms, especially on the exophthalmos and the other ophthalmic symptoms, as well as on the tremors, and the disturbances of the central nervous system." The pamphlet is mostly made up of records of isolated cases and conclusions

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therefrom, and all I can say of isolated cases is that conclusions drawn from them are of no value whatever. The physician must use a remedy in a number of consecutive cases before he can come to a proper conclusion about its value, and my opinion of "antithyroidin," from watching its effects in a number of cases, is that it is practically inert.

Our next patient is a middle-aged woman, and I would draw your attention to the fact that she has been treated on somewhat different lines. Another method of treatment of Graves' disease which has been employed in recent years is the application of X rays. You know the remarkable effect of X-ray treatment in some cases of enlarged spleen, and I thought one might possibly get a similar effect on the goitre in Graves' disease to what one sees in the case of the spleen in splenic leucocythæmia. This patient and some others have been treated for me by Dr. Greg in the Electrical Department of St. Thomas's Hospital. The patient is æt. 51 years, and her history is that at Christmas, 1902, she had an attack of influenza which was accompanied by violent diarrhœa. After the influenza she was troubled with her heart. In June, 1903, her heart became much worse, and a swelling appeared in her neck, which gradually increased in size. She lost flesh considerably, so that she was reduced to a weight of 4 st. 11 lb. On admission to the hospital she was described as a small, thin woman, complaining of palpitation and a growth in the neck for the last three months. On examination her eyes were seen to be staring and prominent, and there was widening of the palpebral fissure. Von Graefe's sign was not present, the sclerotic being quite covered by the lids above, and there was no Stellwag's sign. She had, when she came into the hospital in September, 1903, slight tremor. There was a short first sound at the apex followed by a systolic murmur. In the pulmonary area there was a very loud rough murmur which could be traced up to and could be heard over the thyroid itself. In the neck there was a large goitre of firm consistence, and the neck measured twelve and three-eighth inches round its widest part. During her stay in the hospital she altered little, but she lost some weight and the thyroid remained practically unaltered in size. She was seen again on October 21st, 1904. The exophthalmos was unaltered but the right lobe of the thyroid had increased in size. A loud systolic bruit was heard over it. The heart was rapid and there was a systolic murmur, not conducted outwards, heard with great intensity, as before, at the pulmonary area. The pulse-rate was 136. She was treated at that time with syrup of hydriodic acid. In cases where there is a large goitre in Graves' disease I usually employ some preparation of iodine. It is generally taught that iodine is contra-indicated in this disease, but if the gland is large I do not hesitate to give it. Iodide of potassium or iodide of sodium is useful, but I more often employ the syrup of hydriodic acid or iodipin. Iodipin is a stable compound of iodine and sesame oil. It is prepared in two strengths: 25 per cent. for external use, and 10 per cent. for internal use. In cases where there is a large thyroid I make use of iodipin both externally and internally. I order from 10 ♏ to 30 ♏ of the 25 per cent. solution to be rubbed into the skin over the thyroid gland, and administer the 10 per cent. solution internally in doses of from ʒj to ʒiij daily. If patients have an objection to taking the oil the 25 per cent. iodipin can be combined with malt extract, in the proportion of 1 to 4. Of this mixture one to two teaspoonfuls may be given twice daily. This patient was treated at that time with the syrup of hydriodic acid, ʒj three times a day, but the goitre did not alter in size. Her pulse remained at from 136 to 108, and her weight, which on September 7th was 4 st. 12 lb., on October 22nd was 4 st. 10 lb. During this year she has been treated it the X-ray Department, and has had applications once or twice a week. The result has been that her goitre has slightly gone down, she has put on weight, having increased from 5 st. 2¼ lb. to 5 st. 10 lb. But her pulse-rate remains just as rapid as it was before, and her general symptoms are unaltered. Lately we have also been giving her rodagen, and I am not sure how far any improvement which has been effected may be due to the fact that she is having rodagen, and how far it is due to her having had X rays.

The man I shall show you now presents a very good contrast to the last case. The last one was a woman in whom there was a large chronic goitre and the eye symptoms in her were not very well marked. But this is a man, past middle age, in whom the eye symptoms are very marked indeed, but in whom there is little or no enlarge-

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ment of the thyroid gland. He is æt. 60 years, and his occupation is that of house-painter, and he is a native of Bloomsbury. This man noticed in June, 1905, that his eyes were becoming more prominent and that his sight was beginning to fail. There was no history of any worry or shock, and it is said that, apart from having had painter's colic twice about twenty years ago, he had been in very good health until that time. At first he felt fairly well, but in August his hands became so tremulous that he was unable to carry on his work properly and had to give it up. He attended at the Royal Eye Hospital and was given some medicine. He however, did not improve, his eyes became more prominent, and occasionally he saw double and felt afraid to cross the road. There has been no palpitation, but sometimes he has had a sense of depression over his heart. Two months ago he came to the Ophthalmic Department of St. Thomas's Hospital, where he attended for a month and was then admitted to the ophthalmic ward, from which, after five weeks, he was transferred to my ward. Since he has been in the hospital his sight has been better, and the eyes appear slightly less prominent. He has been getting thin since the beginning of the illness. He perspires at night, especially during the last few weeks. The right eye is now slightly more prominent than the left, and the conjunctivæ are a good deal injected. Von Graefe's sign and Stellwag's sign are both present. As you can see the sclerotic is visible between the iris and the upper lid, due to the widening of the palpebral fissure and more or less immobility of the eyelids on reflex stimulation. The face is pale and somewhat sallow, the tongue clean. There is no visible or palpable enlargement of the thyroid, and I would remind you that one frequently sees the existence of Graves' disease, especially in elderly subjects, without any enlargement of the thyroid. It is one of the peculiarities of the disease that its intensity does not appear to be in any way proportional to the size of the thyroid. You see some cases in which the thyroid is very large, the signs of Graves' disease slight, and the malady runs a favourable course; and other cases in which there is little or no enlargement of the thyroid, the signs of the disease are severe, and the patient is fortunate if she escapes with her life. With regard to the circulatory system, you will notice that there is visible pulsation in the veins of the neck. The apex beat is forcible in the fifth space. The first sound at the apex is loud, and there is a systolic murmur, not conducted outwards into the axilla, with maximum intensity one inch internal to the nipple in the fifth space. The pulmonary second sound is not accentuated, and the sounds at the aortic cartilage are natural. Here, again, I think the murmur is hæmic or exocardial. The pulse is 110, regular, tension rather low, arteries thickened. The temperature has been normal throughout, and the skin is usually moist. The mental condition seems to be quite good. We treated him as an in-patient from January 20th to March 11th, and his condition and weight remained stationary, the latter being 6 st. 8½ lb. But since leaving the hospital we have treated him with rodagen, 30 grains a day, and his weight has gone up to 7 st. 5 lb., and his general condition has, I think, improved. His pulse is now 100, and he says he feels better.

Here is another man the subject of Graves' disease. He has been under treatment since August 18th and is still under treatment. He is æt. 35 years and 5 months. Before he came to the hospital he was passed as a first class life for assurance, so that we may take it there were no obvious signs of disease then. Soon after that he had some troublesome business worries and the symptoms of the disease seem to have come on rapidly. He soon had to give up work, because he was getting very weak and shaky, and sweated profusely after slight exertion. Later his friends called his attention to the marked protrusion of his eyes and swelling of the neck. When he came into the hospital in August he was described as a pale, nervous-looking man, with prominent eyes, shaking hands, and a swelling of the neck. On examination the eyes present von Graefe's and Stellwag's signs and some lateral nystagmus. His hands are very tremulous. The neck is the seat of bilateral enlargement of the thyroid. The cardiac sounds are well heard, the heart's action is rapid and regular, and no murmurs have been detected. On August 18th he was started on rodagen, thirty grains three times a day, and it was also ordered that he was to be out on the balcony during the day-time. And he has taken for the last few weeks ten minims of 10 per cent. iodopin three times a day. He has gained 1 st. 1 lb. in weight, and the pulse-rate, instead of being 124 is about 88. The eyes are not so prominent, and the swelling of

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the neck, he says, has gone down fully an inch. He feels better altogether. The results in this case are quite good, his goitre has become less, he has gained weight, and he is less tremulous.

This case reminds me that I ought to tell you something about the diet of the patients you have seen. In thinking out the diet best suited to patients with Graves' disease, I came to the conclusion that if there is any virtue in rodagen prepared as we have seen from the milk of goats deprived of the thyroid glands it was not judicious to be giving it, and, at the same time, large quantities of milk and butcher's meat from animals with the thyroid gland in full functional activity. Rodagen—which is called the active constituent of the milk of thyroidectomised goats—ought to be counteracted if one is giving large quantities of milk from animals with a thyroid. So for the most part I have allowed milk to these patients only in small quantities. I have also excluded meat from the diet for the same reason. Accordingly the patients whom I have been treating lately have not had milk, at any rate in any quantity, and they have had no beef, mutton, or veal. I have allowed them all kinds of fish, fat bacon, chicken and eggs, vegetables, salads and fruit, cream, butter, bread, and carbohydrates generally. Under that diet they seem to do very well, and as far as I can see it is both theoretically and practically a good one.

The next patient, F. C—, æt. 22 years, is by occupation a factory porter. He had typhoid fever at nine years of age and at about the same period began to have epileptic fits. He attended the out-patient department of St. Thomas's for fits for two periods of three months in 1899 and 1900. He says that for the last two years he has been very nervous, excitable, and easily upset, for some time he has felt very weak, and in the last few weeks it has been observed that his eyes were becoming more prominent. During the cold weather prior to admission his sleep had been much disturbed by a sensation of choking. But there was no difficulty in breathing or swallowing in the day-time. He followed his ordinary occupation up to the time of his admission to the hospital. He perspires freely, especially at night, and complains of occasional pains starting in the epigastric region and shooting down his legs. It was not until he came into the hospital that it was noticed that he had enlargement of the thyroid. At first the circumference of his neck was 18¾ in., now it is only 14¼, so that it has gone down a good deal. The eyes are very prominent, more so than in any of the other cases I have shown you. You can see the sclerotic all round, and Von Graefe's sign and Stellwag's sign are extremely well marked; there is slight lateral nystagmus. The thyroid gland is uniformly enlarged. There is distinct pulsation in the neck, chiefly on the right side. A thrill can be felt over the thyroid and on auscultation there is a blowing systolic murmur. A fine tremor can be seen in the hands. The heart beats rapidly and forcibly, but it is regular, and there is no heart murmur to be heard. The pulse-rate is 120 to the minute. The patient, you will notice, is quite bald, and as you know thinning of the hair is a common event in this disease. But he has had complete alopecia since he was nine years of age, and it seems to have nothing to do with his present disease. Since he has been in the hospital he has kept at about the same weight. His weight increased for a time, but then went down again. Whereas he was 8 st. 11 lb., he is now 8 st. 12¼ lb. He has had ordinary diet, and I do not know whether on that account he has not made the same improvement as the last man I showed you who is in the same ward and on special diet. This man while on full diet has had rodagen, 30 gr. three times a day, and in other respects similar treatment and for the same space of time as the other man, except that he does not go on the balcony, because he is subject to epileptic fits. The man you saw before has had the great advantage of abundant fresh air, having been out on the balcony most of the day, which I think is a very important part of the treatment of Graves' disease. Patients with this malady cannot have too much fresh air.

In conclusion, I may repeat that I consider the diet is an important part of the treatment, and I recommend for most cases plenty of fruit, vegetables, cream, bread, butter, eggs, and but very little meat. I have given you my impressions of anti-thyroidin and rodagen; I think rodagen does some good, although you must not expect a very great deal from it. I think you can give it fairly freely without fear of doing harm, and I should not hesitate in a bad case to give two teaspoonfuls twice a day, while in an ordinary case such as this we give half a teaspoonful three times a day. In a case of Graves' disease with much enlarged thyroid gland we may usefully give the iodides or hydriodic acid or iodipin externally or internally. I must say that of all the remedies I have used in treating goitre iodipin is the most successful, and in such a case as that of the woman I showed you I should expect more from internal treatment by iodipin than from any other remedy. I think the X rays are a little disappointing. One expected a good deal from them, but the results are not very encouraging. I have seen a goitre steadily increase in size whilst under treatment with the X rays.

October 28th, 1907.

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TWO LECTURES
ON
LEPROSY.

Delivered at St. Thomas's Hospital.

By F. M. SANDWITH, M.D., F.R.C.P.,
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LECTURE II.

PRACTICALLY speaking you can divide the various cases of leprosy into nodular, maculo-anæsthetic, and mixed, which of course means both of them. The nodular form always becomes mixed if the patient lives long enough. You see in many of these pictures that the person is suffering from both the nodular and anæsthetic varieties, for sometimes they have obvious lumps on their face and hands, and you can also see that the little finger is crooked, showing that the ulnar nerve is affected. "Lepra mutilans" begins with this condition of the finger and ends with lopping off of the gangrenous portions. It is quite rare to find the anæsthetic form become mixed. So anyone who has nodular leprosy if he lives long enough will have the mixed form, but if he has the anæsthetic form it usually remains as that form. There are exceptions to that rule. There is one difference between these two forms which is of importance to the race, namely that in the nodular form there is first of all an increase of sexual desire and power, and that is followed by atrophy of the testicles and impotence in adults. And the women, on their side, bear very few or no children, and the children who are born to them generally die early. That is to say the nodular form, which becomes "mixed" in time, prevents the sexes from propagating their kind in large numbers. In the anæsthetic form the sexual power is retained for many years, and therefore these people go on reproducing their kind. The onset of both forms is quite irregular. You have certain things which people call premonitory symptoms, but they are not usually recognised until afterwards when one harks back to them and questions the patient. There may be a high temperature at night, a few rigors, drowsiness, dyspepsia, diarrhœa, headache, vertigo, epistaxis (fairly common), and sometimes there is profuse perspiration. You may say at once that those are symptoms which would not lead you to suspect any special disease, and they are slight symptoms and may be entirely absent, so there is not much to be said about them. Certainly I defy you to recognize leprosy in an individual from such very vague phenomena as these. Nodular leprosy has the skin lesion which we call a leproma. It begins first of all with infiltration of the skin, not necessarily a small lump but a patch with infiltration of the skin, generally with hyperæsthesia at first and anæsthesia later. The skin early affected may be on the lobes of the ears, the alæ of the nose, the malar prominences of the cheek, the forehead very often, and the eyebrows. Later on the infiltration appears on the limbs, such as the forearm and the thigh, and sometimes the buttocks. I want to lay some stress on the early appearances, because when you see a fairly advanced case for the first time it is no good telling him he has leprosy—he knows that perfectly and all his friends know it. You ought to try and find out leprosy quite early, before he or his friends suspect it, and that is a little difficult. But those parts of the body which I have mentioned are where you should look for it first. In those situations in a leper you get a feeling of shottiness under the skin, even before you are certain about any infiltration or nodules. And if you have studied leprosy and you know what to look for you are able to recognize the disease six months before other people can. The lesions which we are discussing usually come out in crops. They often begin as papules or as erythematous macules, and they may be single or they may be in groups. Again, they may persist where they originally appeared, or they may disappear, leaving depressions, or breaking down to form a small ulcer covered with a little discharge. The actual nodules I think you will grasp very much better in the photographs than from any description of mine. I would only remind you that it is difficult to get a leper sufficiently early to photograph. For instance, these comparatively early cases of which I have photographs are all about two years old. The nodules are there, but they have not yet begun to ulcerate. Such a person may begin by telling you that he has been ill six months, but if you question him or his relatives you will find that he has had spots on the exposed parts of his body for two

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years. I pass round a photograph of a fairly early case from Ceylon. You will observe there are marks on the face, but, as far as we can see by the photograph, not on the body. You see these marks on the chin, nose, cheeks, and some on the forehead. I also pass round a photograph of an early anæsthetic case, a stage long before the ulnar nerves are affected and long before there is anæsthesia and before the fingers begin to crook. I want to impress upon you the importance of recognizing the disease before it becomes apparent to ordinary people. Here is a photograph at a stage of the illness at which the patient himself would not be able to recognize it, and which the unskilled doctor might not be able to diagnose, because it is so early. These macular patches generally begin in an erythematous form. I have here photographs of the *main en griffe*, the claw-like hand which is produced by the disease, and there are one or two others in which neuritis was a marked feature. In the macular form the erythematous patches are more or less symmetrical, though the area of one side is not necessarily of the same size as that of the other. It often affects the face, and the extensor aspects of the hands, arms and feet quite commonly. It spreads at the periphery and becomes yellowish brown in a white skinned person in the centre of the patch. There is a sort of sub-variety in which you get pigmented patches which sometimes clear up in the centre, and some of them leave little white specks, not of great importance. I want to impress upon you the extraordinarily great area of skin which is involved. Here is a rough sketch which I prepared to show three things : First of all the normal part of the body which is left blank in the sketch, that is to say I could not find anything the matter in that part. Then there is represented the partial anæsthesia which you find in these patches ; it is of the maculo-anæsthetic variety. And lastly, the areas of complete anæsthesia are shaded. In any of those latter areas you could have stuck a knife without causing any pain. But he had no lepromata, nor any hyperæsthesia, because the stage for that had long passed. If there is as much anæsthesia as shown in this diagram you will know that the case is an advanced one. I do not want you to think that every patient who has the anæsthetic variety has as large a surface as that anæsthetic, but often if you carefully question him and test him for an hour you will find that he has a larger anæsthetic area than you or he suspected. It is doubtful if leprosy bacilli are actually present in these patches which I have shown you, or whether they only occur in the nerves which supply the distribution of skin in these areas. Of course there are bacilli present in the body producing it ; probably the trunks of the nerves have them, but not actually the skin. If you cut off a nodule—and as the man has several hundreds he can generally spare you one—you will find the bacillus there, and if you cut away a portion of the skin of the anæsthetic type you probably will not find the bacillus. But if you could take out a bit of his ulnar nerve you would find the leprosy bacillus there ; in the anæsthetic type the bacillus is not found in any superficial place. Now, besides the skin there are various secondary disturbances or lesions which occur. For instance, the hair falls out from the eyebrows, and if a man had a moustache he loses it. First of all the hair becomes very thin, and then almost disappears, only a few straggling hairs remaining. But the hair on his eyebrows may come back when he improves. If he is a man who has much hair on his hands he soon loses that. But the hair does not disappear from his head, and that only means that the skin of his scalp is not turned into a mass of lepromata, and therefore the hair there remains all right. In the other parts of the body the sweat-glands get blocked and compress the hair-follicles, so that they cease to perform their function in nourishing the hair. The nails become brittle, but do not come off. There is atrophy of the skin and sometimes perforating ulcer of the foot. Here are two X-ray photographs showing atrophy of bones. This is the photograph of an adult, and yet if you look at it you will see that the fingers are not only curved but the bones themselves are extremely small.

Now as to the eye lesions. First of all there will probably be infiltration of the conjunctiva with a blood-shot appearance, and the cornea is involved. Later on the iris and sometimes deeper portions of the eye are implicated. At all events, you will readily see that if the conjunctiva, cornea and iris are reduced to a sort of smudge the patient does not see very much. He becomes blind, generally in one eye first, but eventually both. And the anæsthetic patients also become blind, but that is not from a direct infiltration from the outside of

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the eye, but rather from a lesion of the fifth or seventh nerve. Again, the nerves of the eye may be affected just like the arm nerves. Then with regard to the nose, taking the most convenient mucous membrane and the one the most often affected, there is a thickening of mucous membrane there, and snuffling and an odd phonation. Besides that you know, and if you do not know I want to impress it clearly upon you, that the mucous membrane when it is affected will yield you a supply of bacilli. And when you are in doubt about the diagnosis of a case, especially if it is a nodular case, all you have to do is to scrape the inside of the nose, taking away some of the discharge to examine it. Try discharge first, and if you do not find any bacilli in that scrape the mucous membrane and then you will probably find the bacillus. Of course there are other ways of producing the discharge, and one is to give the man a few large doses of iodide of potassium, a drachm to the dose, and that will give him some running at the nose. The larynx is affected very often indeed, and that gives the patient a hoarse voice, and the nerves there also are involved. There is a thickening of the nerves from the presence of the bacilli, and certain nerves are more likely to be affected than others. The ulnar is very commonly affected indeed, so commonly that when you visit the asylum, seeing a hundred cases a day, you feel the ulnar nerve, as most people feel the radial pulse when they come across a patient. I think you will generally find that in the anæsthetic variety the ulnar nerve is increased in size. It is still open to discussion as to how far it is enlarged, just as physicians will differ as to whether and how much a knee-jerk is increased. Sometimes this nerve feels three times the normal thickness. The sciatic nerve also is affected, and the internal popliteal, the radial, and the median, with or without the original erythematous macules. I have told you that first of all there is hyperæsthesia, and that eventually the whole area becomes anæsthetic. The claw-like hand which you find means that there is some paralysis due to the presence of bacilli in the nerves of the dorsal interossei. In the same way there will be an affection of the toe muscles which becomes evident when the patient walks. The bacilli are not present in the muscles, but in the nerves. And as I have already mentioned, you get not only ulcerations of the skin but bone lesions, giving rise to the *lepra mutilans*. Beggars may often be seen exhibiting stumps only, in a miserable condition, but they do not die even when they are as bad as that. In the early stages of the disease there is profuse sweating, but later when the glands get choked up there is an absence of sweating, and hair falls out. I have already mentioned that there is first of all an increased sexual desire, and then a loss of it, and I have some figures dealing with sterility. At one leper asylum in eighteen years there were 2864 lepers, and only 26 children were alive at the end of that time, though the lepers were allowed to marry and two of these children had become lepers. Of course one should have said under the heading of prevention that the children ought to be taken away from leper parents, and not kept in asylums. There is one other symptom which I should mention, because it is very disagreeable to the nose of the average person, and that is the stinking odour which comes from these people when they are ulcerating in the nose, palate and pharynx. You cannot look at an advanced case of leprosy without being very sorry for the individual, but I defy you to remain long indoors in his presence if he has had no treatment and the whole of the mucous membrane of the nose is ulcerating.

Now I want to say a few words about the morbid anatomy. You can readily guess what the skin is like. You find lesions and ulcerations of the larynx, and if you look at the tongue, pharynx, and respiratory passages, generally you find large numbers of lesions. You will also examine the nerves, especially those I have mentioned, the ulnar, median, radial, tibial, peroneal, facial and popliteal. You will find some glands affected but only secondarily to the skin affection, for instance the femoral, cervical, and some others. You will find bacilli in internal organs, such as the liver, spleen, testicles and kidney. I do not suppose any of you need to be convinced that leprosy is not a skin disease merely, but you will see from what I have said that internal organs are also affected. The bacilli have also been found in less frequent sites, such as in the ovaries, the intestinal wall, the mesenteric and lumbar glands, and in the bone marrow. Those are the rarer places where it can be met with. In one of my cases the median nerve at the wrist and the posterior tibial nerve at

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the ankle were three times their normal diameter after death. The supra-orbital nerves are very much increased in size. The result of inoculation of the bacilli into animals is negative. But to that dogmatic statement I must make one exception, because Nicolle has succeeded in inoculating leprosy under the skin of the ear of two monkeys. It has often been tried by others, who failed, but this particular observer appears to have succeeded. On the sixty-second day nodules appeared and two weeks later bacilli were found in them. After several weeks some small nodules appeared, which contained the leprosy bacilli inside mononuclear leucocytes. There is a disease in rats which is called leprosy, but probably it is not identical with the condition which we are talking about.

Now as to the *diagnosis* of the disease. It is not difficult to diagnose except in the very early cases, and in the maculo-anæsthetic form. Remember to examine the eyebrows, the lobes of the ears, the buttocks, for thickening of the skin in patches, and tiny nodules which infiltrate. Repeated epistaxis is a suspicious sign, but if you find thickened nodules or irregular patches on the face or on the limbs you will examine to see if they are anæsthetic. Also examine the tip of the little finger, because anæsthesia appears there very early. Then examine other parts of the body. The ulnar nerve is specially picked out by the disease, I do not know why. Look at the tip of the little finger and the parts which the ulnar nerve supplies in that region, and eventually you may find the whole area of distribution of the ulnar nerve anæsthetic. In all cases be sure to examine the nasal secretion, either natural, or after doses of iodide of potassium, or by scraping the nasal mucous membrane. When in doubt examine one of the lepromata and you may find the bacillus in it. Syphilis ought not to confuse you in comparison with leprosy, although you must remember if you go abroad to tropical countries that you will see much worse syphilis than anything in England. In the tropics a man with syphilis is treated because he has a chancre and he does not know the difference between the different sores; he is a unicist. I daresay you hardly remember that in the old days it was thought that gonorrhœa and soft chancre and syphilis were all one disease, and I am now going rather farther back than the time when I sat on those benches. Still, the tropical patient has not yet awoke to the fact that there is a difference between a local chancre and a constitutional disease like syphilis. He comes for treatment because he has a chancre, and when that seems better he goes off and does not seem to want anything more. In consequence there are very bad tertiaries to be seen in the tropics, such as you may read of in books but do not see in this country, or at all events in such a hospital as this. And you must remember that a man may have both syphilis and leprosy. In such a case you put him upon mercury and his syphilis will be benefited by that, whereas the leprosy will not be greatly improved by it. Then with regard to leucodermia—a very definite disease, especially on a dark skin. In the tropics leucodermia is a very much more definite thing to look at than here in the out-patient room. It has nothing to do with leprosy, and you know that in leucodermia there is no loss of sensation, whereas in these macules of leprosy there is anæsthesia. Moreover the leprosy macules are not really white, but of a greyish colour, while the leucodermia patches are entirely free from pigment. There is another disease also, ainhum, a troublesome affection in which the little toe looks as if a constricting band had been put round it and it was practically dropping off in consequence. That is not leprosy, and has no connection with it, but the two things may occur in the same patient, as I have seen in patients in the tropics. With regard to the diagnosis between the two, if you are in doubt you will examine under the microscope, and in ainhum you will find no leprosy bacilli. Syringomyelia has been mistaken for leprosy but it ought not to be.

Now as to *prognosis*. It is quite doubtful if there are any real cures of the disease, but you must not say that to intelligent lepers, because it makes them inclined to suicide. Many cases of leprosy have been arrested, which is, of course, the next best thing to cure. Some of the gloomiest people will tell you that tuberculosis is not curable, and many people in France to-day say that syphilis is not curable. But you will be able to point out cases of syphilis and tubercle which have been cured. And one can point to cases of leprosy which have been arrested in their progress, so that the disease has not been found to go any further in them. The nodular form of leprosy lasts from five to ten years; that is the average. You will see

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that the photographs of some of these patients describe them as having been inmates in the asylum for twenty years. But the anæsthetic form may last fifteen to thirty years; it is of very much slower growth. The man with the anæsthetic form is not so unsightly nor so troublesome to his neighbours, nor so odorous. In Trinidad there is a report published of 109 post-mortems. Thirty per cent. of these lepers had tubercle, whereas of the non-lepers under the same circumstances only 18·5 per cent. had tubercle. This means that lepers were almost twice as liable to tuberculosis as the non-lepers. There is a close similarity between the bacilli of the two diseases, and it is not surprising that patients with leprosy should have some sort of affinity for tuberculosis. But there are other things too—poverty, bad hygiene, depression of spirits—which predispose the leper to tuberculosis. Another interesting and even more important fact is that in the same number of post-mortems it was found that 32 per cent. of the lepers had nephritis post mortem, whereas of non-lepers only 7·5 per cent. had it. So that lepers suffered four times more frequently from nephritis than other people. And in connection with that do not forget the condition of the skin, the infiltrated patches at the beginning and afterwards extending to large areas. And if the skin cannot act very well, of course more work is thrown on the kidneys, and they consequently suffer and become chronically diseased. I ought to have mentioned that in the mixed form of the disease albuminuria is one of the things which you look for when the nephritis is beginning.

As to *treatment*, I will begin by telling you the worst treatment as far as I know, and that is the Egyptian. This plan is to apply the actual cautery, both to the nodules and in circles round the affected skin to try and prevent the disease from spreading. I have pictures showing that among the collection on the table. If a patient comes to a native barber with leprous nodules on the hand, the barber will put a red hot circle round, and when the patient finds an extension of the nodules and comes back to the barber he will put another mark round higher up. Many lepers seem to get a sort of hanging pouch of skin over the olecranon. This treatment by the cautery is absolutely valueless, but the idea underlying the treatment is that there is a sort of devil entering the skin, and it is necessary to take care that it does not get to the vital organs of the body. Surgery is of some use in leprosy, but the actual cautery is of none. When the larynx is invaded and there is dyspnœa you will find sometimes that tracheotomy, in an advanced case, will prolong life. General and local treatment, on general principles, are of course important; that means hygiene, good food and so on, and an open-air life, in fact treatment like that for tubercular patients. And almost any treatment of the skin helps them too. For instance, sulphur baths have been found useful, probably by helping the skin to act better and taking some pressure off the over-worked kidneys. Tuberculin has often been tried in large doses. The reaction of tuberculin is delayed and lasts longer than in the case of tuberculosis, and though some cases get worse a few have apparently got better. One has seen it tried in twenty cases at a time, and sometimes you may think there is a great improvement, though in others there is no improvement at all. X rays are only useful in quite early cases, where there is only an early involvement of the skin. Chaulmoogra oil you may give in almost any form, provided the patient can tolerate it. Begin by giving it by the mouth, and if you find the stomach does not tolerate it give it in capsules, or in an emulsion with gum. Begin with 20 ♏ in the day and go up, if your patient will allow you, to 300 ♏ in the day, increasing the dose gradually. But unfortunately the stomach rebels in time. You may give it by the rectum with milk, you may rub it into the body twice a day, and you may give it in sterilized tubes intra-muscularly. This last method sometimes causes painful lumps and eruptions, and if you do not take care an abscess forms. But if antiseptic precautions have been taken and the oil itself is pure the local trouble will not be septic, and you will be as safe with this as with any other form of intra-muscular injection. Most drugs are useless, but they are good if they improve the general health of the patient, and sometimes mercury has been found to help, just as it does in the case of old syphilis, not in clearing up the disease but in acting as a general tonic. You advise the leper to leave the infected country if possible, and give him warm clothing, because he hates cold weather, and is very liable to disease of the kidney.

Now about *prevention*. I think leprosy ought

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certainly to be notifiable, like any other communicable disease, and if there is a suspicion of any insects carrying the disease as we hinted at last time you must either use mosquito nets in hot weather or protect the community from insects which might come from the leper. Naturally you would not use the same laundry as that used by the lepers. It is necessary to burn all dressings from the leper's sores, to use rags or paper handkerchiefs for the discharges from the nose, because they contain so many of the bacilli; and of course you will carry out disinfection of the clothing. And if you had the power you would isolate all cases which had open sores or a nasal discharge. But it is not logical to lock up all these people for life, as is done. It is more logical to lock them up so long as they have a running sore or bacilli in the nose. When leprosy becomes arrested from treatment or from the efforts of nature I think the patient should go through an intermediate period of quarantine, and then be allowed his liberty. Personally, I do not think they should be allowed to propagate their species, but if they do propagate, their children should be separated from the leprous parents. There are certain trades and professions which lepers should be debarred from, such as selling milk, or handling fish, or bread, or indeed any eatables whatever. Here is the picture of an Egyptian clerk who had an interesting history. He was attacked with mixed leprosy, the nodular and the anæsthetic form, when he was fifteen years of age. He said there had been cases of leprosy in his village, but never in his family. Five years after he was attacked, that is to say when he was twenty years of age, during one of the temporary lulls in the disease, he thought he was cured, and he then married. His disease appeared again, and he had a wretched time, becoming extremely ill. There was this unfortunate odour which I have told you of, his wife left him, and he got such an anæsthetic condition of the nerves of his right arm that he could no longer write, so that he was dismissed from his post. You can imagine his miserable condition, for he had lost his wife, his work, and his friends. When he had been ill ten years he came under the notice of Dr. Tourtoulis, a Greek colleague of mine in Cairo, who found that the patient had lost his eyebrows and moustache and had a hideous appearance. He was treated with these intra-muscular injections of Chaulmoogra oil. It was first given by the stomach, but the patient said he would rather have the leprosy. The face was covered with leprous nodules, some of them beginning to break down, his breath was stinking and the soft palate was ulcerating. His fingers were becoming atrophied, and he had claw-like hands and deep perforating ulcers on the sole of the right foot, and in the dorsum of the left foot. There was also evidence of double broncho-pneumonia, and a spasmodic laryngeal cough. After fifty intra-muscular injections, each of one drachm, he was so improved in general condition that there was a complete disappearance of the ulcers on the feet. After continuing the same treatment three months longer the hair began to grow again on his eyebrows and upper lip, and the sweat-glands, which had been inactive, began to secrete again, and the face and limbs gradually improved. The treatment went on for over five years. During that time some 544 injections were made, amounting to about 96 ounces of Chaulmoogra oil—nearly 5 pints. He not only improved extremely in appearance and his hair came back, but he regained his looks, for he was once a very handsome man. He got a new wife, and became manager of the bank in the place where he lived, and even represented the British as their Vice-Consul.

October 28th, 1907.

Rectal Anæsthesia.—J. H. Cunningham believes that this method of inducing narcosis may be employed to produce anæsthesia preliminary to the administration of ether by the mouth, especially if the patient is known to take ether badly. He has devised a special apparatus to overcome rectal disturbances and to control the degree of anæsthesia. In his own cases there had been no diarrhœa or bloody stools. In a few cases temporary colicky pains and painful abdominal distension have been present. Details of administration cannot be discussed here. He believes that the lung eliminates ether as fast as the rectum absorbs it, so that danger is lessened. Comparatively little ether is required. There is no stage of excitement. Vomiting is infrequent. Excess of secretion is reduced to a minimum. Recovery is rapid.—*Medical Record*, vol. lxxii, No. 13.

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THE TREATMENT OF SURGICAL EMERGENCIES.

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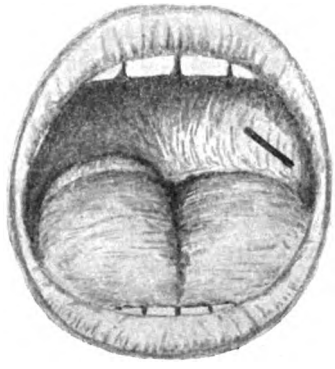
Acute tonsillitis and peritonsillitis—Retro-pharyngeal abscess—Injuries to respiratory centre—Cut throat—Transfusion—Laryngotomy and tracheotomy.

In *acute tonsillitis and peritonsillitis* it not infrequently happens that an abscess forms which fails to evacuate itself and requires surgical assistance. It is most important to bear in mind the situation where the pus usually forms; this is not in the tonsil but above it, in the cellular tissue of the soft palate. It frequently happens that when you attempt to see the back of the patient's mouth there is considerable difficulty in doing so. The teeth can only be separated a short distance, and most of the soft palate is hidden by the tongue; saliva may also obstruct the view, because of the difficulty the patient has had in getting rid of it, owing to the pain caused by attempts at its expulsion. In these cases it is advisable to give the patient about half a tumblerful of hot carbolic lotion 1 in 100, to which a teaspoonful of bicarbonate of soda has been added, and instruct him to wash the mouth out with it and to hold some of it in the mouth for a minute or two, with the head inclined in such a way as to allow the lotion to come in contact with the affected part. This cleanses the mouth and at the same time acts as a local anæsthetic. The patient should then be seated in a chair facing a good light and means taken to prevent his drawing his head back at the moment when you attempt to incise the abscess. This can be managed by seating him in a chair with a high back against which the head rests, or by placing an ordinary chair against the wall, so that the wall, protected by a cushion, supports the head, or if this does not suit the light an assistant may stand behind the chair and support the patient's head with both hands. The incision of the abscess does not appear to cause much pain, but if thought advisable a little 10 per cent. cocaine solution may be applied to the soft palate on the affected side by means of a little cotton wool held in forceps. A straight, sharp, rather narrow-bladed knife should be taken and the blade wrapped round with sticking-plaster to within about three quarters of an inch of the point. On now looking into the mouth and gently depressing the tongue with a spatula and moving the patient's head to get the light to the back of the mouth, probably the lower margin of the soft palate and the margin of the anterior pillar of the fauces on the affected side will be out of view, being forced downwards by the swelling and hidden behind the tongue. The soft palate, however, can usually, with a little care, be seen to be red and bulging. The most prominent part of this swelling is where the incision should be made. The knife should be introduced with its edge towards the middle line and should cut upwards and inwards for about half an inch. The incision will generally be about half an inch deep. If further exploration is necessary it can easily be done by introducing closed sinus forceps into the wound to the necessary depth and then opening them.



The dark line shows the situation of the incision for opening a "quinsy."

This sketch shows the usual position for the incision. If you could see the margin of the soft parts concerned you would probably see that the incision started rather below the upper end of the anterior pillar of the fauces and about one third of an inch external to its inner edge and passed upwards and inwards in the soft palate. It has been suggested that for safety and to avoid alarming the patients Hilton's method of opening these abscesses should be employed, by passing in a closed pair of sinus forceps and then opening them without the aid of the knife at all; but it seems to me that this must be more painful

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than the use of a sharp knife. We get a number of these cases sent across from the medical side with a request to open the abscess; they are all treated in the way I have described, by incision with a knife, with success and without difficulty. Many physicians regard acute tonsillitis as a rheumatic manifestation, and consider that by keeping the patient in bed and giving salicylates and alkalies, and the general treatment for rheumatic fever, that formidable affection is often warded off.

Retro-pharyngeal abscess.—This affection may be met with in two forms, either acute or tuberculous. The former is seen in small children and probably starts in the lymphatic glands found in childhood, which lie between the pharynx and the prevertebral aponeurosis, about the level of the second cervical vertebra. The tuberculous variety may start in the same glands or be connected with tuberculous disease of the cervical vertebræ. I think if time and circumstances permit and the child can take an anæsthetic that both varieties are better opened from the side of the neck, behind the sterno-mastoid muscle, though it must be confessed that most cases of the acute abscess do well when opened through the mouth, but this should never be done if the abscess is tuberculous. Children are in considerable danger of being suffocated, drowned in their own pus, if these abscesses burst spontaneously, and this should be borne in mind when examining these cases with a finger in the mouth. This should be done with the greatest gentleness, and if rupture occurs the child should immediately be held head and face downwards so that the pus can escape by the mouth instead of flooding the larynx. I have known this accident happen during examination by an eminent surgeon and with fatal results. These abscesses generally form elongated swellings which push the pharynx forwards; they are placed to one side of the middle line, and there may be a swelling on both sides; if the swelling is centrally situated it will probably be low down. The reason why these abscesses appear laterally above, in the part behind the naso-pharynx, is that in that situation the median raphé of the pharynx is attached behind to the prevertebral aponeurosis, and divides the retro-pharyngeal space into two compartments. In acute cases the abscess may be so large and the dyspnœa so marked that you do not care to incise it through the mouth or to give an anæsthetic. The danger of suffocating the child with pus after incising the abscess can generally be avoided by laying it on its side with the head lower than the rest of the body. A straight small-bladed knife should be taken and the blade guarded to within one third of an inch of the point with sticking-plaster. The end of the blade should be laid flat on the pulp of the left index finger, which is then passed along to the fluctuating swelling and the necessary vertical incision made down its centre. This can easily be done by touch as you may not be able to see the parts. When the abscess is opened the child should be held so that the pus runs forwards out of the mouth. Or most of the pus may be first removed by an aspirating syringe by passing the hollow needle guided by the index finger back into the abscess in the same way as the knife; the abscess wall can then be incised with little risk of pus passing downwards. As these cases generally occur in babies of one or two years old they are too young to cleanse the mouth and throat themselves. It is therefore advisable to have the throat swabbed out frequently with some non-poisonous antiseptic. In dealing with a tuberculous abscess, the symptoms of which are not so acute as those just described, an anæsthetic should be given and an incision an inch or more long made behind the upper part of the sterno-mastoid muscle, which is pulled forwards with a retractor. The transverse processes of the cervical vertebræ are then felt for and a director pushed along close to the anterior surface of one of them into the abscess. Your left index finger in the patient's mouth and touching the abscess will assist you in guiding the director. As soon as pus appears in the groove forceps are slipped in and opened vertically to make the opening large enough to admit the finger, when the condition present, such as the presence of a sequestrum, can be ascertained. The abscess cavity should be well irrigated out and rubbed with swabs held in forceps and then closed without drainage after all *débris* has been removed. If the case is one of caries of the vertebræ the head and neck should be fixed in an apparatus suitable for the treatment of that disease. Extreme care must be taken not to move the diseased part of the spine while the child is unconscious. The abscess may fill again and require a repetition of the treatment.

Injuries to respiratory centre.—Some years ago I

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heard Sir Victor Horsley deliver a lecture in which he described an experiment in which a sheep had been shot through the head with a rifle. The respiratory curve and the pulse tracing of the animal were being recorded mechanically at the time. At the moment of injury the diagrams showed that respiration ceased instantly, but that the heart tracing, after some irregularity, went on as before, showing that the heart, after being pulled up for a short time, started again spontaneously and continued beating. If nothing were now done for the sheep the heart in time ceased beating and the animal was dead. But if artificial respiration were performed for a short time respiration again became spontaneous, and the sheep lived until respiration and circulation again ceased owing to the hæmorrhage caused by the injury and consequent compression of the brain. The lesson to be learned from this is, that the respiratory centre succumbs to an injury more easily than the circulatory centre, and that artificial respiration done in time, before the heart stops, may save life. Some years ago a man who had fallen from a scaffold was brought into the hospital quite unconscious and apparently dead; he showed no signs of respiration and no pulse could be felt at the wrists. In fact a very experienced casualty nurse asked me to go at once and see him as she thought he was dead. His pupils were considerably and equally dilated, but not quite as widely as after death, and I thought I could feel a flutter with my hand over the heart. Without moving him from the ambulance or moving him from the view of a number of other patients, artificial respiration was at once performed, and someone else gave a hypodermic injection of strychnine and ether. After a time signs of spontaneous respiration appeared, but soon passed off when the artificial respiration was stopped; it was therefore continued, and eventually respiration continued normally. The man lived, and one day, as I was going round the ward, he thanked me for saving his live, as someone had told him the story of the artificial respiration, etc. I cannot help thinking that many lives might be saved in this way, by artificially keeping on the patient's respiration till the respiratory centre has had time to recover from the shock which had inhibited its activity.

Cut throat.—Fortunately in cases which do not die within a few minutes the large blood-vessels in the neck are not usually injured, and in many cases the air-passages are not opened. The first thing to do in such a case is to stop any bleeding; first by digital pressure if necessary, then by ligature of the bleeding points, and after thoroughly irrigating the wound with an antiseptic solution, such as 1 in 2000 perchloride of mercury, the divided structures should be sewn up in layers with buried sutures of iodised catgut and the wound closed without drainage. If possible the irrigation should be done before the ligatures are applied, as there is then less risk of sepsis. If any large vessels are divided they will have to be controlled and both ends found and ligatured. If the air-passages are injured the wound in them should be closed with catgut, preferably lightly chromicised, and if tracheotomy has not been thought necessary it may be wise to insert a drain-tube to avoid the risk of surgical emphysema. If there is embarrassment of respiration tracheotomy will have to be done at once and the tracheal wound held open to allow blood and clots to be coughed out and the air-way cleared. A tracheotomy tube should then be inserted and the trachea packed above it with a long strip of gauze, to prevent more blood trickling into the bronchi, or, if available, a Hahn's or Trendelenburg's tube can be used, either of which will block the trachea round the tube. If gauze is used it must be secured to prevent it being drawn into the trachea by the patient's inspiratory efforts, and antiseptics used to disinfect the wound must not be allowed to run into the the trachea. When the air-passages are not injured many cases do perfectly well without tracheotomy, the wound being sewn up in layers and closed without drainage. The patient should be propped up as much as convenient in bed with the neck bent forwards to relieve the tension on the stitches.

Transfusion.—If much blood has been lost it may be necessary to transfuse the patient with saline solution, of the strength of one teaspoonful of common salt to a pint of water. The median basilic vein is the one usually chosen for this purpose, but, if it is large enough, the median cephalic is preferable, as it does not lie over the brachial artery as is the case with the former vessel, and there is, therefore, less risk of injuring the artery, also the internal cutaneous nerve may be injured if the median basilic is chosen—this has been known to cause painful spasm of the muscles of the upper limb. A tape should be tied round the arm

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to make the veins prominent. Where they are very collapsed they are easier found through a transverse incision. A double thread is passed under the vein and the lower one tied. It is easier to get the cannula into the vein if it is only partly divided; the upper thread is then tied, holding the cannula in position. The fluid must be run in slowly, and if there are any signs of embarrassment of respiration, or restlessness, or twitching, the flow of fluid must be suspended for a time, as these symptoms show that the blood passing through the lung contains too much salt solution. Up to four pints may be injected if necessary; in bad cases transfusion may have to be done a second time. Some advise that adrenalin chloride 1 in 100,000 should be administered with the salt solution to diminish shock. The tape round the arm must, of course, be removed when the cannula is in the vein and the fluid ready to flow in. It is best to use an indiarubber pipe and funnel for this purpose, not a syringe, and great care must be taken that no air is injected; this is done by seeing that the apparatus is partly filled with fluid before and after introduction of the cannula. The temperature of the fluid as it runs into the vein must be 100° F. or slightly over.

Laryngotomy and tracheotomy.—Laryngotomy is quickly performed by pinching up the skin of the neck in a vertical direction over the crico-thyroid membrane and transfixing it as this makes a transverse incision. A pair of curved, sharp-pointed scissors is now taken and the point thrust through the crico-thyroid membrane and the handles forced apart; in this manner an air-way is made in a few seconds.

Tracheotomy is done through a vertical incision through the skin over the upper part of the trachea and commencing over the crico-thyroid membrane. The point of the knife is then passed in the middle line between the two sterno-hyoid and sterno-thyroid muscles, which are separated and drawn from the middle line. A heavy clip forceps may be attached to the edge of the muscles each side, and allowed to hang, one on each side of the neck, and will act as retractors. A transverse incision is now made along the lower edge of the cricoid cartilage, dividing the cervical fascia attached in that situation, and with a blunt hook the fascia is dragged downwards, taking with it the veins and the isthmus of the thyroid gland, leaving the trachea quite bare. By this means much troublesome bleeding is avoided. A sharp hook is then inserted into the lower margin of the cricoid cartilage in the middle line and held in the left hand, and the upper two or three rings of the trachea are divided in the middle line, the edge of the knife pointing towards the chin. Or a sharp hook may be passed round a ring of the trachea, one on each side; then when the trachea is opened traction on the hooks will hold the wound open. If no sharp hook is at hand the trachea must be steadied as well as possible by the fingers of the left hand, and when it has been opened one side of the wound is depressed by the tip of the index finger of the left hand and the tube slipped in over the dorsum of the finger-nail. It is very easy to push the tube by the side of the trachea instead of into it, when it will be noticed that the respiration is not properly relieved. If one sharp hook is inserted into the side of the trachea the latter is in danger of being rotated, and the air-tube may then be opened at the side instead of in the middle line.

October 28th, 1907.

HIGH-FREQUENCY CURRENTS: THEIR PRODUCTION, PHYSICAL PROPERTIES, PHYSIOLOGICAL EFFECTS, AND THERAPEUTICAL USES. By H. EVELYN CROOK, M.B., B.S.Lond., F.R.C.S. Eng. London: Baillière, Tindall and Cox.

Dr. Crook, in his preface to this volume, "hopes that it may in some way help to bring before the notice of medical men the undoubted therapeutical value of high-frequency currents in certain pathological conditions." We welcome this addition to medical literature, and trust it may result in placing treatment by high-frequency currents in the hands of properly qualified medical practitioners. Special attention has been given in this work to the therapeutical uses of high-frequency currents, and it is this portion of the volume that will afford most help in the every-day work of the general practitioner. A remarkable account is given of the successful treatment of some cases of chronic psoriasis by Dr. Somerville, and encouraging reports are given in detail of instances of disease of the nervous system in which the application of the high-frequency currents has effected most gratifying results. A record is given of a patient who complained of symptoms pointing to debility, anæmia, and a condition of the system simulating slight attacks of Raynaud's disease. High-frequency currents were administered irregularly during a period of three months, after which time the health of the patient was much improved.

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ANGINA PECTORIS.

A Lecture delivered at the London School of Clinical Medicine, Seamen's Hospital, Greenwich.

By GUTHRIE RANKIN, M.D., F.R.C.P.Edin.

THERE is probably no ailment in the whole nosology of disease which so fills the lay mind with alarm, and the professional mind with a sense of responsibility, as angina pectoris. The very name is synonymous, in the popular conception of the disease, with sudden death, and even the accurate and discriminating knowledge of the skilled observer compels the admission that the life of him who has experienced a true anginal paroxysm hangs by a thread which strains to the breaking point under every influence engendering emotional disturbance or physical exertion.

Angina pectoris was first accurately described by Heberden; subsequently its dependence upon disease of the coronary arteries was demonstrated by Edward Jenner; and at a still later date Nothnagel established the fact that the characteristic phenomena of the condition are capable of being produced by functional conditions as well as by organic changes.

It is now universally admitted that angina may be either true or false, and that, though its manifestations are closely similar whatever the cause be on which the attack depends, yet the gravity of their significance can only be estimated on a careful consideration of all the circumstances. The prognosis is much more hopeful in some cases than in others, but it always demands expression in terms of guarded reserve; because though that variety of the disorder which we are able unhesitatingly to classify as *true* is attended by the constant contingency of sudden death, the variety which is recognised as *false* is not by any means so unimportant that the possibility of serious eventualities can be definitely excluded.

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True angina is a disease of middle or late adult life, and is met with, in the great majority of cases, in men. Almost without exception it occurs in those who suffer from sclerotic changes in the arterial system. It is not surprising therefore that gout, rheumatism, and diabetes are important predisponents; nor that alcoholism and syphilis are revealed in the history of a notable proportion of cases. It is an uncommon complication of mitral disease, but in aortic disease it constitutes a constant source of uncertainty and danger. Though extremely rare, attacks of true angina have been recorded in children who are the victims of congenital heart disease. Haubner and Swoboda have each recorded a fatal case occurring in an infant under eighteen months old.

False angina may occur at any age. It may arise either from neurotic or from toxic causes. The neurotic type of the affection is mostly met with in women, and is induced by emotional disturbance, fright, flatulent dyspepsia, or neurasthenia. The toxic type, especially that dependent upon the excessive use of tobacco, is most frequent in men.

In all cases of angina pectoris it is usual to find a hereditary tendency to instability of the nervous system; and in all varieties of the disease the circumstances of life prove to be such as induce undue strain on the cardio-vascular mechanism, acting either from behind through the ventricular walls, the coronary arteries, or the aorta; or in front from narrowing of vessels consequent on arterial degeneration or vaso-motor constriction. The exciting cause of the attack is almost invariably a sudden increase of blood-pressure, which may be produced by excitement, exertion, sudden exposure to cold, or excessive distension of the stomach. Excitement is the most frequent proximate cause of false angina, while physical exertion is the most common antecedent of the graver variety. Exposure to cold is a fertile source of both forms of anginal disturbance. There can be little doubt that angina is the true explanation of the "cramp" which attacks and frequently leads to the drowning of swimmers who plunge headlong into cold water.

As regards the pathology of the disease many views have been held. The one outstanding fact is that, in fatal cases the coronary arteries are found almost without exception to be practically occluded either throughout their length, or at their orifices, by endarteritis affecting the root of the aorta. Sclerosis of the coronary arteries is very common, and is frequently found after death in patients who have presented no anginal symptoms during life. The false variety of angina, which is supposed to be due to a functional contraction of the peripheral vessels, is said never to prove fatal, and therefore it cannot have any pathology, but the fact that death does not frequently happen in cases of this type is no proof that the symptoms are not produced by similar changes to those which are accountable for the true variety of the disease, only occurring in a very early stage and under conditions of organic vitality sufficient to provide for recovery. It is easy to understand how neurasthenia, auto-intoxications, emotional strain, and many neurotic conditions may cause cardiac embarrassment by maintaining a constant excess of contraction in the peripheral vessels, because, as Gaskell has pointed out, the cardiac, like other muscles, may become, under the influence of fatigue, cramped from the accumulation of waste products. If the heart and vessels are sound, this condition of fatigue is automatically overcome by the influence of the depressor nerve of Ludwig, which, being stimulated, inhibits sympathetic activity and relieves pressure by causing relaxation of the arterioles; but any failure in Nature's mechanism of adjustment, from whatever cause produced, must obviously involve grave risks. William Russell, of Edinburgh, contends that the common factor in every variety of angina is hyper-sensitiveness of the vaso-motor centre in the medulla, and that, to a large extent, this hyper-sensitiveness is induced by the action of poisons, such as tobacco, alcohol, and excessive proteid food. He points out that an arterial reflex having its origin in the abdomen is a normal arrangement for adapting the circulation to the processes of digestion. During active digestion, contraction or hypertonus of the systemic arteries always occurs, and the reflex is accentuated by high feeding and alcoholic liquors. This natural reflex is abnormally sensitive in some individuals, and when it becomes associated with increased tension it is frequently the direct cause of arterial sclerosis. He declares that after the arteries have become sclerosed they are particularly sensitive and specially liable to hypertonic contraction. This

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attractive theory, if proved to be true, offers a solution of many difficulties and reduces the proximate cause of the disease, in whatever form it is met with, to a single and simple explanation. In true angina the forms of heart disease which are usually met with as explanatory causes are fatty or fibroid degeneration of the myocardium, usually secondary to coronary disease; valvular disease, especially aortic regurgitation; and atheromatous or aneurysmal changes in the aorta.

The precise cause of the pain which is such a prominent feature of all anginal attacks has given rise to many theories. Of these the more important are: (1) That it is due to a neuralgia of the cardiac nerves; (2) that it is due to a cramp of certain areas of the cardiac muscle; (3) that it is due to increase of intra-ventricular pressure with consequent stretching and tension of the nerves in the muscular substance; (4) that it is due to ischæmia of the myocardium from temporary failure of the coronary blood-supply. Broadbent says truly that the condition of the heart-walls must be the dominant factor in angina, else the disorder would be much more common than it is in Bright's disease, where arterial tension reaches its maximum.

Tessier, in a recent thesis, also accepts this view, and has endeavoured to prove that, whatever the primary cause of the disease may be, the painful paroxysm is always due to cardiac distension. He says: "If we scrutinise the attacks of anginal pain, which are of frequent occurrence in aortic insufficiency, either spontaneously or following an effort, we find that in the majority of cases they coincide with acute dilatation of the left ventricle, that is to say, with an increase of the previously existing dilatation. True angina pectoris almost invariably presents by way of anatomical substratum a lesion of the coronary arteries, which determines more or less narrowing, hence the ischæmia of the myocardium. It is open to us to believe that this inadequate arterial irrigation of the myocardium determines, under the influence of stress or emotion, sudden cardiac insufficiency and immediate distension, causing irritation of the nerve terminals of the cardiac plexus which is the cause of the crisis."

If this theory be accepted as reasonable in its application to cases of true angina, it is not inconceivable that in the false variety of the disease a similar sequence of events may be responsible for the pain, the condition of the coronary arteries being probably one of functional spasm instead of atheromatous change.

The salient features of true angina pectoris are familiar to us all. The initial attack usually supervenes in the daytime, and is provoked by sudden exertion or violent emotion, either of which induces a rapid increase of arterial tension. Subsequent attacks may occur spontaneously, and often happen during the night. The patient, most frequently a man of over forty years of age, is suddenly seized with agonising pain in the præcordial region, which is usually accompanied by a sense of constriction as though the chest were gripped by an iron hand. The pain radiates in several directions, especially towards the left shoulder, down the arm, and along the tract of the ulnar nerve to the ring and little fingers. It may also extend to the right shoulder, or towards the cervical plexus in the neck, but it rarely takes a downward direction. The areas of the skin in which the pain is felt generally show no objective phenomena, but Gasne and Chiray recently reported a case to the Société Medical des Hôpitaux de Paris in which, after anginal attacks with pain radiating down the left arm, there subsequently developed along the internal surface of the upper arm a linear series of four patches of lichen planus corresponding to the distribution of the intercosto-humeral nerve. The pain is accompanied by a feeling of intense anguish and an overpowering sensation of impending death. The patient is compelled to cease from any effort in which he may be engaged; he suddenly halts in the standing or sitting position, whichever he chances to occupy at the moment, and seeks support from any object that may be at hand. His face becomes pallid, bedewed with cold perspiration, and not infrequently the lips are cyanotic. The breathing is short and difficult, and an imperious desire for air is experienced. The pulse becomes small, hard, and sometimes irregular, and the heart sounds are feeble but not necessarily abnormal, though frequently they assume the fœtal rhythm. The duration of the attack is short, and varies from a few seconds to three or four minutes. It ends suddenly, and its cessation is followed by eructations, distension of the stomach, salivation, or copious micturition, and a sense of extreme exhaustion. Not infreqnently it culminates in death. In minor attacks the patient may only

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experience a passing sensation of præcordial anxiety without actual pain, or he may suffer from sudden pain down the left arm or in the epigastrium without any sense of constriction in the chest. The painless attacks, which are mostly met with where there is some existing cardiac disease, have been named by Gairdner "Angina sine dolore." They are liable to be recurrent and create in the patient not so much a feeling of anguish as one of oppression, restlessness, and uncertainty. They sometimes terminate fatally from syncope. In false angina, though the main features of the more grave form of the disease are reproduced, there are certain points in which they differ one from the other. Persons of either sex who are of the neurotic temperament may be affected. Women suffer more frequently than men, and are specially liable to be attacked by the disorder at the period of the menopause. Those are most vulnerable who suffer from what is popularly known as "a feeble circulation"; this is manifested by coldness of the extremities, a liability to chilblains, "dead" fingers and toes, chilliness and blueness on exposure to even moderate degrees of cold, etc. The attack mostly occurs under the influence of emotion; it may also be induced by the contact of cold winds when walking in the teeth of them; or it may happen in bed on first getting to sleep, and is then generally attributed to distension of the stomach from an unsuitable evening meal.

Dilatation of the stomach is capable of giving rise to attacks closely resembling the paroxysms of true angina. If the attacks are, from the onset, nocturnal, and particularly if they manifest a periodicity in their recurrence, it may be concluded that they are of gastric origin. The pain and præcordial constriction are similar to those which characterise the true variety of the disease, but do not, as a rule, present themselves in such an acute form. The pulse is, in most instances, rapid and often irregular, and its tension is always high. The heart, though probably excited in action, is not abnormal either to percussion or auscultation, but the second aortic sound is unduly accentuated. The patient complains of faintness, palpitation, a sense of fulness in the epigastrium, and painful difficulty in breathing. She is restless, possessed of a feeling of dread, and impelled to throw open the nearest window that she may get air to relieve the apnœa from which she suffers. Cold perspirations, flatulent eructations, and a copious secretion of pale urine are common sequlæ. Most cases of false angina can be distinguished by the facts that the attack supervenes spontaneously, that it lasts longer than in true angina, that it is accompanied by restless movement, that it tends to recur again and again before finally subsiding, and that there usually co-exists throbbing of the abdominal aorta, mobility of one or other kidney, vaso-motor manifestations, and other evidences of nerve instability. In that form of the disease which depends upon the excessive use of tea or tobacco, the only trustworthy method of arriving at an accurate opinion is to wait and see whether the attacks recur after the use of these substances has been abandoned.

The differential diagnosis between true and false angina is not, as a rule, a matter of great difficulty, but there are cases where it is impossible to arrive at a definite conclusion from the observation of a single attack. The patient must be kept under observation for some time and the state of the heart and nervous system studied both at the time of the attacks and during the intervals between them. It must not be forgotten that attacks, false at first, may ultimately become true, nor must it be overlooked that false attacks, though mostly free from danger, are not always so. In the true variety of the disorder we have to deal with a person who shows evidences of arterial degeneration, frequently accompanied by cardiac hypertrophy. There may be some degree of dilatation of the aortic arch, when the aortic second sound will be exaggerated. Disease of the aortic valves may or may not be present. The existence of a mitral regurgitant murmur in association with anginal attacks is suggestive of a hypertrophied heart which is failing under the strain of excessive arterial pressure, or from myocardial debility sufficient to prevent it coping with the intrinsic pressure to which it is subjected. The earlier attacks usually come on under the influence of exertion, and the patient is always compelled to stop short in the midst of whatever he may chance to be occupied with.

In the false variety the patient may be of any age. Arterial tension is always excessive, but there is no cardiac lesion beyond the accentuation of the second aortic sound, produced by the increased tension. Gouty conditions, constipation, emotional

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instability, gastric disturbance, flushings, and other vaso-motor phenomena are instructive antecedents. The patient is not compelled to stand immovable when the attack comes on, but on the contrary is restless and clamorous for air. The true causes of false angina must be remembered, and inquiry should always be made as to the patient's habits, particularly in regard to tea and tobacco.

The following table is compiled from Huchard's differential scheme :

True angina.	False angina.
(1) Most common between the ages of 40 and 50.	(1) May occur at any age.
(2) More common in men.	(2) More common in women.
(3) Attacks brought on by exertion.	(3) Attacks occur spontaneously.
(4) Attacks rarely periodical or nocturnal.	(4) Attacks often periodical and nocturnal.
(5) Agonising pain and sensation of compression as in a vice.	(5) Pain less severe and sensation of distension.
(6) Absence of vaso-motor phenomena.	(6) Vaso-motor phenomena usual.
(7) Pain of short duration.	(7) Pain may come and go over an hour or two.
(8) Attitude one of silence and immobility.	(8) Attitude one of agitation and activity.
(9) Cause, sclerosis of coronary arteries.	(9) Cause, neuralgia of nerves and cardiac plexus.
(10) Prognosis grave.	(10) Prognosis favourable.
(11) Result often fatal.	(11) Result never fatal.

The only disorders with which angina is at all likely to be confounded are hepatic or renal colic, gastralgia, or muscular neuralgia of the chest-walls, but to the careful observer no difficulty can ever arise in arriving at a correct opinion. The prognosis in true angina is always serious, particularly so in cases where the symptoms are of primary cardiac origin. When the attack is dependent upon vaso-motor spasm, even when the heart is hypertrophied or the seat of valvular disease, there is room for relief under proper treatment, and with care future attacks may be guarded against. In every case where the arteries show evidence of atheromatous degeneration and where there is, therefore, reason to fear the existence of sclerosis in the coronary vessels, no guarantee can be given that life may not terminate suddenly at any time. In false angina, on the other hand, the prognosis is, in the great majority of instances, favourable, but death does sometimes occur in the course of the paroxysmal attack, so that the forecast ought never to be made too absolute. Moreover, the high arterial tension which accompanies false angina is liable to become permanent, in which case it ultimately induces cardiac hypertrophy, and this, in its turn, may be responsible for the development of a true seizure under the influence of sudden and unusual mental or physical stress.

The question of treatment must be considered under two aspects : that which is necessary during the attacks, and that which is called for in the intervals between them. In view of the intensity of the pain and the urgency of the symptoms, the attack demands prompt measures for its relief. The only drugs upon which reliance is to be placed for this purpose are : morphia, oxygen, nitrite of amyl, and chloroform. Morphia should be administered hypodermically in a dose of a quarter of a grain, and its efficacy is increased by combining it with one hundredth of a grain of atropine. Morphia not only relieves the pain of the attack, but it also asserts a powerful dilating action on the small vessels by which peripheral resistance is reduced. It should not be used in cases where there is a threatening of cardiac failure or where pulmonary œdema is present. Under such circumstances ether or caffeine, administered hypodermically, are useful substitutes. But morphia has the disadvantage of taking a short time to exert its influence. In the first instance, therefore, it is always wise to administer by inhalation five or six drops of nitrite of amyl, the value of which as a vaso-dilator was first discovered in 1867 by Lauder Brunton. Its action is rapid but fugitive. In a few seconds the face becomes flushed, the eyes injected, and a sense of throbbing is experienced in the head. The effect of the drug does not last more than from thirty seconds to a minute, and in many instances the inhalation must be repeated until the morphia has time to manifest its influence. Sometimes it proves quite powerless, and it is under such circumstances that chloroform finds its opportunity; a few whiffs act promptly and seldom fail to give relief. Similarly, the free inhalation of oxygen is often valuable. The oxygen not only relieves the gasping breathing, but it secures the circulation through the constricted coronary arteries of super-oxygenated blood by which the myocardium is powerfully stimulated. It is convenient to recommend those who are

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subject to anginal attacks to carry always with them some of the small glass bulbs provided by manufacturing chemists, each containing the proper dose of amyl nitrite for one inhalation. These are easily broken in a handkerchief at the onset of the attack.

Other useful vaso-dilators are nitro-glycerine and nitrite of sodium, but they are less rapid in their action than those already referred to. To some patients nitrite of amyl is an offensive drug, and for them the best substitute for self-administration is nitro-glycerine made up in one minim doses of the 1 per cent. solution into small tabloids, which may be carried conveniently in the waistcoat pocket. To get the best and speediest effect the tabloid should be nibbled and allowed to dissolve in the mouth before swallowing.

In the intervals between the attacks the patient should, as much as possible, avoid physical fatigue and violent emotions. He ought to have long nights in bed, and the ordinary habits of his daily life should be disciplined so as to promote a quiet and uneventful routine. Frequent holidays, during which there is a complete abstention from the duties of ordinary occupation, are desirable. A moderate amount of quiet exercise in the open air is a daily necessity. The diet should be simple and bland; proteids, tea and coffee must be taken in very moderate amount, and all varieties of food that tend to tax the digestive power are best done without. Pure water, or a natural alkaline water, may be drunk with meals, and a tumblerful of piping hot water sipped in the early morning before breakfast promotes the activity of the portal circulation and the excretion of accumulated waste. Milk should enter largely into the daily regimen. Tessier suggests that salt should be withheld as much as possible. After an attack of angina, Muklen recommends a saltless diet for five days, followed by two days of milk diet before returning to the usual plan of mixed meals. In order to keep the arterial tension low, mild mercurials should be given periodically: 1 gr. of calomel, or 2 gr. of blue pill, combined with 3 gr. of rhubarb may be taken as a matter of routine once a week, and at other times the bowels should be kept gently open by a morning dose of Carlsbad salts. When the tension increases it ought to be controlled by iodide of potassium or sodium in 10 gr. doses, combined with one or two minims of the 1 per cent. solution of nitro-glycerine and three minims of Fowler's solution taken after meals three times a day. This combination may be continued safely over some months or even years, if care is taken to prevent saturation by omitting it for one week out of four. Where the history reveals a gouty element in the case, colchicum may be added to the mixture. In addition to its anti-gouty value, it accentuates the vaso-dilating influence of the other drugs. Tonics are sometimes indicated, and of these among the best is a mixture of dilute hydrochloric or phosphoric acid with strychnine in decoction of bark. In cases where angina is an associate of valvular disease circumstances may arise which call for the exhibition of digitalis. The existence of angina does not contra-indicate the use of this drug, but its constrictive influence on the arterioles should be counteracted by combining it with either nitro-glycerine or erythrol tetranitrate. It is claimed for the latter drug that its influence is more lasting than that of the other vaso-dilators. In the false variety of the disease, treatment must be pursued along similar lines so far as the attacks are concerned. When they are severe nitrite of amyl or even morphia may be of necessity, but milder remedies often suffice. Valerian, sumbul, aromatic spirit of ammonia, or chloric ether given at the onset may be effectual, and to any of them nitro-glycerine should be added if the pulse indicates a high degree of tension. In the intervening periods the clue to successful medicinal treatment lies in ascertaining the determining cause of the seizure. This is not always easy to accomplish, but it is obvious that bromides or hydrobromic acid are likely to prove useful in excitable persons of neurotic temperament, iron or arsenic in anæmic conditions, and simple alkaline or acid mixtures, according to indication, in those who are the victims of disturbed digestion.

In a large proportion of cases, especially those occurring at or about the climacteric, the following prescription, ordered in capsule form, and to be taken three times a day, will be found useful: Valerianate of zinc, three grains; ichthyol, three grains; arsenious acid, one fourtieth of a grain; and extract of cannabis indica, one fourth of a grain.

The bowels must be thoroughly evacuated every day, and for this purpose a pill containing half a grain of calomel, quarter of a grain of aloin, and three grains of compound assafœtida pill, taken at

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bedtime when necessary, will generally prove efficacious.

As in the true variety of the disease, attention to diet, exercise, and habits of life is essential. It is of special importance to reassure the patient by a cheerful prognosis, and to advocate a properly regulated but active interest in the work of the world. Introspection is the curse of many of the sufferers from false angina, and this can only be combated by a well-occupied life devoted to interests outside of self.

Finally, the wise words of Sir Douglas Powell may be quoted as a counsel of perfection: "He who would treat angina pectoris in its multiform degrees with all the success that can be looked for must take the cases in hand on broad lines in accordance with the well-defined principles of medicine, pursuing such lines into such detail as may be appropriate to each case."

November 4th, 1907.

LIVER ABSCESSES: THEIR ETIOLOGY, SYMPTOMS, AND TREATMENT.

A lecture delivered at the Medical Graduates' College and Polyclinic.

By HENRY CURTIS, B.S., F.R.C.S.

SUPPURATIVE hepatitis may be conveniently divided into tropical abscess and non-tropical.

ETIOLOGY.

(*a*) *Exciting Causes.*

Non-tropical abscesses are classified according to their cause, whether they are, as is most common, pyæmic in origin, traumatic, the result of a direct injury, or the presence of a foreign body, such as a pin, migrating from some other part of the alimentary tract—though pins impacted, *e. g.* in the appendix, etc., generally set up pyæmic abscesses. The results of ulceration of gall-stones, parasitic worms, and specific fevers, such as enteric, though rarely, should also be remembered as producing suppuration. These rarer causes are chiefly of importance in that a knowledge of them may enable us to detect hepatic suppuration occurring as an obscure and complicating sequela.

It may surprise some to be told that Mitchell* has recorded thirty-three cases of pins impacted in the appendix, out of which in no less than eight instances there was some form of an intra-hepatic suppuration, in most cases multiple or pylephlebitic abscesses, and Trowbridge mentions a case of a shawl pin found in the appendix, followed by a liver abscess which proved fatal.†

Speaking very broadly, non-tropical abscesses may be ascribed to pyæmia, due to pyogenic micro-organisms, whether the strepto- or staphylococci, the *Bacillus coli*, or the microbes of the specific fevers, coming to, and passing through, the liver circulation, traumatism acting simply by providing an area of lowered resistance, where any circulating organism can find a foothold and so give rise to an abscess.

The majority of tropical abscesses are associated with, and, as we shall see, are believed to be due to, the presence of amœbæ of the kind found in certain forms of dysentery. Omitting for the moment the so-called "idiopathic abscess," where there is a single large abscess, non-dysenteric and non-amœbic, we may therefore distinguish between two main varieties of hepatic suppuration by saying that whilst tropical abscess is of animal parasitic origin, the majority of non-tropical are essentially pyæmic, though tuberculosis and actinomycosis must not be forgotten, and an even more important and more frequent cause probably than these is syphilis, now known to be due to Schaudinn's spirochæte. An instance of mixed infection, where amœbic tropical abscess was probably complicated by a breaking-down gumma, is included among the illustrative cases.

Idiopathic abscess.—It is well to bear in mind however, that in warm climates another variety of the single large abscess is met with, not associated with the presence of amœbæ, and non-dysenteric in origin, according to some of the most careful observers, who state this variety to make 15 to 30 per cent. of their cases.

In a considerable number of cases seen by Davidson, to whose recent writings I am so

* 'Johns Hopkins Hosp. Bull.,' 1899, p. 35. (Quoted in Rolleston's 'Diseases of the Liver.')

† See Editorial in 'Brit. Med. Journ.,' November 12th, 1904, entitled "Visceral Abscesses after Appendicitis."

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largely indebted for these remarks on etiology, the presence of dysentery or diarrhœa could be positively excluded. In idiopathic abscess, also, there is polymorphonuclear infiltration of the liver tissue, whereas in amœbic abscess there is an absence of leucocytic accumulation, either in the necrotic tissue or the capillaries. In the amœbic abscess the process is more purely necrotic, in the idiopathic abscess, it is inflammatory, terminating in an invasion of the degenerated areas by pyogenic bacteria. It is held by some that organisms were present originally, but died out subsequently.

As to the actual or exciting cause of the more common variety of tropical abscess, there is no doubt now that it is directly associated with the dysentery which in so many cases immediately precedes it, and upon the sudden healing of which it may immediately supervene.

It is necessary, however, to distinguish between the two main varieties of dysentery, the bacillary form and the amœbic. The bacillary form is seen in Japan, certain parts of China, Ceylon, Zanzibar, South Africa, British Guiana, etc., the amœbic variety being met with in India, Tonkin and Cochin China, Mauritius, and notably in Egypt, also in Venezuela, amongst other places in South America. The bacillary form of dysentery is also familiar to us here by reason of its recent presence in asylums and institutions in Great Britain and in Europe, where, however, the amœbic variety is not unknown, being, in fact, endemic on the eastern shore of the Adriatic, Dalmatia, Roumania, etc. The dysentery so commonly resulting from the hardships of war will, as Davidson points out, be of that variety prevalent in the country, temperate or tropical, in which warfare has arisen.

Observation has shown that the bacillary form of dysentery is only occasionally followed by hepatic suppuration, and this chiefly where there has been deep ulceration, with sloughing of the bowel, in which case one or several large abscesses may result. This is quite the exception, however, and one may regard bacillary dysentery as only rarely being followed by abscess of the liver, the reverse being true in the case of the amœbic variety. In the bacillary dysentery of Japan, to quote Rolleston,* Shiga has never seen hepatic abscess as a complication, and the rarity of hepatic abscess after asylum dysentery in England, and in warfare carried on in temperate climates, is well established.

Further reference will be made to this subject under the heading of "Climate."

Alone, or associated with pyogenic organisms, the amœba of dysentery is the cause of the common type of tropical abscess. Davidson estimates that only about one twelfth of the cases of amœbic dysentery go on to abscess, and we have to consider what other factor is necessary for its development.

Normally, the liver has the power of destroying both toxins and microbes of a dangerous nature brought to it from the intestines. This function is impaired when the vitality of the liver is reduced for any reason. The first stage is that, during an attack of dysentery the products of amœbic activity in the intestine proceed to the liver by the portal circulation, setting up necrosis of the liver cells immediately around the central vein. This may produce symptoms of slight hepatitis, which recur from time to time, and lower the resisting power of the liver to subsequent microbic invasion, that is to say by the amœbæ themselves, with or without associated pyogenic organisms. It is easy to understand how much more readily the liver will succumb to the attack if it has been previously crippled by alcoholism, malaria, and other causes of congestion and degeneration. In other words, for the development of hepatic abscess we require a liver unable to perform its functions normally; given hepatic inadequacy, the amœbæ of dysentery will do the rest. Apart from hepatic insufficiency it is rare, though perhaps not impossible, for either amœbæ or other microbes to set up suppuration in the liver.

Mode of invasion.—The amœbæ reach the liver generally through the portal circulation, but in the case of superficial abscesses, at least, Rogers favours the view of transmission *viâ* the peritoneum from, more especially, the hepatic flexure of the colon.

(*b*) *Disposing Causes.*

(1) *Climate.* — In spite of its name, tropical abscess is curiously limited in its distribution, especially in comparison with that of the two diseases ordinarily associated with it, dysentery and malaria. The climates of India and Tonkin, and Egypt, Mauritius, and Venezuela, appear to be amongst those most adapted for the development

* *Ibid.*

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of such abscesses, whilst in certain parts of China, *e.g.* Hong Kong, and in South Africa, strange to say, where dysentery is endemic, liver abscess is comparatively rare. The geographical distribution of the two different varieties of dysentery, previously discussed, accounts for this difference, assuming the direct and indirect evil effects of the various tropical and sub-tropical climates on the individual to be about equal.

During the Boer War (1899–1901) inquiries made from medical officers by Rolleston* when in Africa in 1901, showed that single hepatic abscess, as a sequela of South African dysentery, which was not amœbic, was extremely rare, and out of 716 cases of dysentery treated in two Yeomanry Hospitals, (Deelfontein and Pretoria), there were only two cases of single hepatic abscess, and two fatal cases of pylephlebitis. The same appears to be true of Rhodesia. In the 'Bulawayo Hospital Reports' from 1898 to 1906, omitting two years, the records of which are not available, the total number of patients was as follows :

As to the 9 liver abscesses amongst the whites there were 7 deaths, the present writer being fortunate enough to have charge of the two successfully operated on in 1904, including one associated with appendicitis and gumma of the liver.

The report for 1897–8 incidentally alludes to certain liver abscesses operated on during the year, but no number or result is stated.

Taking the numbers available, 9 abscesses in 5536 cases amongst the whites, we must conclude that the percentage of liver abscesses is very low and would, in normal years, probably have been even lower, for 5 out of the 9 cases occurred during the Boer war (1899–1901), when so many troops, for the relief of Mafeking, passed through Bulawayo, from Beira and Salisbury; the 5 abscesses, with 5 deaths, occurring amongst a total number of whites admitted—2579—including 1321 malarias with 28 deaths, and 133 cases of dysentery with 14 deaths. Possibly, too, some of these soldier patients had come from India and thus brought with them the amœbic type of dysentery.

Summary of Table Comparing Incidence of Liver Abscess in Bulawayo, Rhodesia, with that of Malaria and Dysentery.

Total No. of cases admitted during six years.		Malaria (including black-water fever).				Dysentery.				Liver abscess.			
		Whites.		Natives.		Whites.		Natives.		Whites.		Natives.	
Whites.	Natives.	Adm.	Died.	Adm.	Died.	Adm.	Died.	Adm.	Died.	Adm.	Died.	Adm.	Died.
5536	2625	2805	67	731	57	270	24	152	57	9	7	2	1

Thus, out of 5536 whites, during a period of six years, only 9 cases of liver abscess occurred, in spite of the fact that 2805 suffered from malaria, with 67 deaths (including blackwater fever cases), and 270 from dysentery with 24 deaths.

Amongst the natives, who numbered 2625, in six years there were two cases, one dying from general tuberculosis of the lung after operation, so that possibly the liver disease was also tuberculous; the second being a case of multiple pyæmic abscesses from septic pylephlebitis, associated with cirrhosis of the liver, and hæmatemesis. At the autopsies the appearance of the pylorus and pancreas suggested to some the presence of malignant disease, but there was considerable doubt as to the nature of the condition observed.

* *Ibid.*

The influence of climate, including mean temperature, altitude, etc., is, therefore, related to its effects on the geographical distribution of the fauna and flora of disease, and contributes to the disease in question, directly by favouring the development of the specific organism or parasite, and, equally importantly, by lowering the vitality or resisting power of the host, human or animal, eventually becoming its victim.

Sex.—Men are far more liable than women, children being but rarely affected. The reasons for the greater susceptibility of men than women, living otherwise under similar climatic conditions, are said to be: (1) Greater exposure to sun and hardships generally; (2) greater liability to traumatism; (3) greater tendency to alcoholism. The good rule prevalent among all intelligent Europeans

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in the east, to be strictly temperate and never to drink until sundown, is thus shown to have a sound scientific foundation. However, in South Africa, where such a rule is less stringently adopted, liver abscess is decidedly rare, this apparently depending on the different organism causing the dysentery prevalent.

Age.—The disease is most commonly met with among young adults from twenty to forty-five and upwards. But one case of seventy-five years of age is recorded, as well as a series of sixteen tropical abscesses amongst children, and two young children where no organism could be found in the abscess.

Habits.—Alcoholism predisposes to hepatitis in both white and coloured people, and such a condition, by lowering the vitality of the liver, may easily lead on to suppuration. The same is true of exposure to hardships of various kinds, great heat, etc. Manson has emphasised the need for caution in the case of young officials, civilians, and others, proceeding to hot climates where the temptation to self-indulgence in diet, drink, etc., is so great.

The hepatic insufficiency resulting from exposure to heat, fatigue, and chill, or from a diet excessive for the needs of life in a hot climate, alcoholism, and malaria, etc., allows of easy invasion by the amœba of dysentery.

Race.—White races suffer more than coloured in Africa, and the same is true in India, Europeans suffering considerably more than Easterns, though the reverse is true in the case of dysentery in both places, though the actual hospital admissions may sometimes be misleading. This difference is related to the different habits of the two races, alcoholic natives in India losing their immunity in this respect.

Symptoms and duration.—The onset is often very insidious. In many cases, however, the date of onset of more or less acute symptoms can be given. Examination of the records of the cases I have examined shows the duration to have been from ten to twenty days up to eighteen months, a history of a month to six weeks being common. In some cases the history may extend to ten years, or even more, especially where a patient, long resident in a tropical country, has arrived in Europe in the winter.

The patient is generally anæmic and wasted, obviously suffering from malaise and cachexia. He occupies the dorsal decubitus, as turning to one or other side, especially to the left, often accentuates the feeling of weight, or the dragging sensation to which he is subject. This is undoubtedly worse where painful adhesions have formed. The appetite is generally poor, the tongue is coated with a dirty brown fur, the sides and tip being sometimes of a bright red colour. Jaundice, common in pyæmic abscesses, is rarely present in tropical abscess unless there is pressure on the bile-ducts. Vomiting, stated by some to be frequent, in many cases is absent. It is more marked in the rare cases of the abscess pointing downwards (see Case 4). If early and severe, and associated with chills, or rigors, and high fever, as sometimes, but very occasionally, happens, liver abscess may readily be mistaken for a bilious remittent type of malaria familiar to all who have lived in fever districts.

In the majority of cases *the stools* are loose, and of a light yellow colour. In some cases diarrhœa is actually present, or on the other hand, more rarely, constipation, requiring enemata throughout the treatment. When, in the absence of blood and mucus, such as is met with in typical dysentery, there is frequent slight diarrhœa and marked tenesmus, the amœbæ of dysentery may be sometimes found in obscure cases on examining film preparations of the dejecta. *The urine* is scanty and high-coloured, and may contain urobilin and albumen.

Cough and expectoration.—Frequently there is distressing cough, in the early stage simply from irritation of the phrenic nerve, later, probably, from perihepatitis and secondary diaphragmatic pleurisy. Pain in the chest from pushing up of the diaphragm by the abscess or from slight basal pneumonia and pleurisy, with signs of consolidation, crepitant râles and friction, is not uncommon. It is most often on the right side, its presence on the left probably indicating suppuration in the left lobe. If the abscess has burst into the lung the sputum becomes tinged with a light fawn or brick-red colour, and amœbæ may be detected therein even before actual rupture according to some, and as a premonitory sign of it.

Heart.—The heart's apex beat is occasionally displaced upward in the mammary line, as high as the fourth space, Dymott, who notes this, regarding this displacement of the heart's apex beat upwards

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as of very considerable value where the diagnosis otherwise is rather obscure. Under such circumstances the left lobe of the liver would probably more often be the seat of suppuration.

The cardinal symptoms of liver abscess, however, are fever, pain and tenderness over the liver, pain being also referred in a certain proportion of cases to the right shoulder, enlargement of the liver, and profuse night sweats, with the onset of hectic fever at night.

Fever.—The temperature on admission may be found raised, 101° to 103°F. The pulse is weak and rapid, 90 to 120, or more, per minute; respirations, 24 to 40 per minute. The temperature chart indicates a continuous fever, with or without remissions, but with the onset of definite suppuration hectic fever is set up, and when this is associated with profuse night sweats the diagnosis of hepatic abscess is practically certain, given a painful, tender, enlarged liver. The fever, however, is sometimes absent throughout, or for long periods, and is often very irregular. This is borne out by the two charts I show you, relating to Cases 1 and 4 of the illustrative series.

Perspiration.—Night sweats are frequently very profuse, but may be absent. They appear to be more frequent in the acute than in the chronic varieties. When present, especially when associated with an up-and-down temperature, they form a very useful sign, as indicating acute suppuration. In Case 1 night sweats were entirely absent, whereas in Case 4 they were so profuse as sometimes to require changes of clothing six times in the night.

Pain.—Pain in the shoulder is not always present, and its value is discounted by the fact that it may occur with hepatitis only. Though generally referred to the right shoulder blade it is sometimes located to the left, apparently when the abscess is in the left lobe of the liver.

Pain and tenderness over the liver.—As a rule this is located to the right, front or back, or the right side of the chest, but may be referred to the left in disease of the left lobe. Tenderness is best detected by exploring the distended intercostal spaces all over the region of the liver with the finger tip.

Condition of the abdomen; enlargement of the liver.—This may be so slight as not to be perceptible at first. There may be little or no enlargement, as in Cases 1 and 4, or it may be completely obscured, as in Case 3, by ascites, and only on diminution of the ascites, spontaneously or on tapping, is the enlargement revealed. Not infrequently the lower part of the right side of the chest is distended, the intercostal spaces being widened or bulged out. Where an abscess points in front of the liver the enlargement may be very marked, the lower border reaching to the level of the umbilicus, or even to that of the iliac fossa. A definite ovoid tumour with the long axis vertical is sometimes seen in the epigastric and hypochondriac regions, moving with respiration, unless there are firm adhesions. Where the abscess forms in the upper and posterior part of the liver, the enlargement is generally in an upward direction, this being detected by percussion. Where the abscess is approaching the surface and is about to point there is redness and œdema of the skin over the side—right, left, or front of the chest—to which the abscess is approaching.

Symptoms connected with the lung and pleura.—The lung is more often ruptured into than the pleural cavity. Where the abscess has escaped into the chest the physical signs are those of purulent effusion. When the lung is involved amœbæ have, as already mentioned, sometimes been found in the sputum as a premonitory sign. The pus from the liver abscess soon makes its way into a large bronchus, and typical liver pus, variously compared with "chocolate" or "anchovy paste," is seen in the expectoration.

Symptoms connected with the rupture of the abscess into other regions.—The pericardium, the peritoneum, the stomach, the colon, and the kidney have been the seats of such ruptures, and the symptoms will vary accordingly. In the case of the pericardium there is sudden acute pain and the signs of pericardial effusion. There are not many cases on record, one reported by Jacobsen,* of Havana, being due to abscess in the posterior part of the left lobe of the liver. In the case of the peritoneum there will be acute peritonitis, and so on.

Diagnosis.—The principal points have already been enumerated under the heading of "Symptoms." A hectic temperature, accompanied by profuse night sweats, with a tender, enlarged liver, occurring in a patient who has lived in a hot climate, or

* 'Brit. Med. Journ.,' 1889.

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has suffered, or is suffering, from dysentery, should lead one to suspect abscess. Exploratory puncture will clear up the diagnosis in the majority of cases, the needle being inserted as a rule through the tender spot or spots (see also under "Treatment").

The finding of polymorphonuclear leucocytosis has in some cases led to the discovery of hepatic abscess in an otherwise obscure case, which would thus apparently be of the idiopathic variety.

Prognosis.—The prognosis must depend on the patient's general condition, and the duration and extent of suppuration. Where the abscess is diagnosed early and can be reached easily and satisfactorily evacuated, the outlook is good, but with long-standing and extensive suppuration in a very cachectic subject, broken down by hardship, or alcoholism, syphilis, etc., the outlook is decidedly grave.

Treatment.—This is prophylactic and operative.

(*a*) *Prophylaxis.*

Consideration of the etiology leads to the conclusion that tropical abscess may be placed in the list of preventible diseases, and every care should, therefore, be taken by the new arrival in a tropical country to live abstemiously, both as to solids and liquids, especially alcohol, remembering that less diet than in a colder climate is required, and that alcoholism definitely destroys the immunity otherwise observed amongst natives in India and other countries where the amœbic form of dysentery is endemic. Exposure to undue fatigue and to chills must always be avoided, and the body kept in as healthy a condition generally as possible by judicious exercise and a regular mode of living. Malaria, another preventible disease, must be guarded against by the use of mosquito netting curtains, and prophylactic doses of quinine at regular intervals, *e.g.* every tenth and eleventh day, and dysentery avoided, as far as may be, by general sanitary measures, including the boiling of water used for drinking and also cooking purposes, *e.g.* for the cleansing of green salads, and the scouring of crockery and other kitchen utensils; otherwise salads, etc., may become potent sources of infection in such countries.

For those who have lived for some time in a hot climate, withdrawal, at least every three years, to a colder climate is most desirable, but the holiday should be planned so that the arrival in England is not at a time when bleak wintry weather is being experienced.

(*b*) *Operative Treatment.*

This should always be arranged for at the time exploratory puncture is decided upon, so that the line of successful puncture may be followed up at once, one anæsthetic then only being required.

In practice, one punctures where the physical signs have enabled the pus to be located. Redness and œdema, with a localised tumour, indicate that the abscess is superficial and pointing, and puncture is directed accordingly.

The seat of the abscess is in the right lobe in 70 to 80 per cent. of the cases, in the left lobe in 5 to 15 per cent., in both lobes in 9 per cent., and in the Spigelian lobe in 2 to 5 per cent., the upper and posterior part of the right lobe being the common seat of abscess, which much less frequently occupies the concave surface (Davidson). Case 4 illustrates an abscess in the last-mentioned position.

If the abscess is evidently pointing, treatment simply consists in following up the exploratory puncture needle, left *in situ*, with a long, narrow-bladed knife, when the abscess cavity is rapidly struck and the pus evacuated. The insertion of a stout, wide bore tube, sutured to the integuments completes the operation. The danger of serious hæmorrhage is practically *nil*.

If there are no signs of pointing the abscess must be sought, Cheyne says, by exploratory puncture, the trocar and cannula of fairly wide bore being thrust through either the eighth or ninth intercostal space in the anterior axillary line; or failing this, through a point in the nipple line just below the right costal margin. If this also gives a negative result the needle is next thrust through the ninth or tenth right intercostal space, vertically below the angle of the scapula. If pus is found the rib may be exposed and a portion excised. This is not always necessary. On deepening the incision, if the parietal pleura is exposed it is dissected up and the layer over the diaphragm separated, so as to be brought into easy apposition with the parietal layer, to which it is then sutured. When this is done, or if the pleural cavity has not been opened, the diaphragm is incised along the line of the cannula, left *in situ*, Cheyne advising that the liver be pushed well up against the diaphragm,

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so as to prevent the escape of pus between it and the liver.

A pair of long-bladed dressing or polypus forceps is now inserted by the side of the trocar, and a drain tube inserted along the track thus dilated up. Gauze packing for twenty-four hours may be advisable where the pleural cavity has been laid open.

Manson has devised a very simple method, details of which are to be found in his book on 'Tropical Diseases,' specially valuable, perhaps, in the case of abscesses deeply situated and difficult to drain.

After puncture with a large trocar and cannula 4 to 5 in. long by ⅜ in. in diameter, the trocar is withdrawn. A correspondingly wide bore drainage tube, closed at its inner end, but having a wide lateral opening made close by, is stretched taut, so as to elongate and narrow its calibre considerably, on a stilette, and in this condition thrust along the cannula to the back of the abscess. The cannula is carefully withdrawn and then the stilette, leaving the now completely contracted drainage tube *in situ*, the return from its stretched to the normal calibre thus entirely obviating any leakage of pus along the track through the diaphragm.

Where it is necessary to open through the abdomen, if the liver is adherent, there is no difficulty in opening the abscess. Where the respiratory movements of the cannula left in place indicate few or no lesions, some operators prefer to insert gauze pads within the peritoneal cavity, with the view of promoting adhesions. Even where no adhesions exist the risk of soiling the peritoneum by the escaping pus does not appear very great, so that preliminary suture of the friable capsule of the liver to the peritoneum has been largely abandoned. It may, however, after inserting temporary pads of gauze, be advisable to evacuate as completely as possible the abscess through a large cannula, before withdrawing it to insert the stout wide-bore drainage tube. In this way the liver is enabled to fall back into a more normal position with regard to the abdominal wall, so that there is less likelihood of leakage, or of kinking of the tube.

Illustrative Cases.

Case 1.—The first case I shall read to you illustrates the single large abscess type, which, when the pus has been evacuated, readily closes. It also illustrates the latency of liver abscess.

The patient was a miner, æt. 45 years, who had lived in Matabeleland for eleven years, having entered with the "1893" Column.

Until a year before he came under my care he had been a heavy drinker. For eighteen months he had been ill, but the symptoms were so obscure that the diagnosis of liver abscess was not entertained by the medical attendant. Insomnia, attributed to nervousness, was troublesome. There were no night sweats. The patient had, however, been able to work until a few days before admission, on which date he complained of pain and a dragging sensation in the region of the liver, worse on turning on to the left side. No enlargement of the liver downwards could be detected on admission nor subsequently, and nothing else abnormal in the abdomen.

On the second day after admission, two points of tenderness were for the first time made out, one located to the eighth right intercostal space, in front, above the level of the gall-bladder, the other tender spot being at the right costal margin in the ninth interspace behind, in the posterior axillary line. Friction was heard over the areas of tenderness.

The upper level of the liver dulness was at the lower border of the fifth rib.

The temperature never went beyond 99° F., and this only on two occasions.

Exploratory puncture, performed on the eighth day after admission, revealed the presence of pus, and the next day an incision through the ninth space, deepened along the track of the re-inserted cannula, was made. A few ounces of creamy, and then blood-stained, pus were evacuated, and found to contain amœbæ. A wide-bore drainage tube three inches long was inserted, and its outer end sutured to the skin.

The drainage tube was removed on the nineteenth day, and six days later the patient left the hospital with the wound practically closed.

Case 2.—The following notes, from the Surgical Report of University College Hospital, 1899, were made by me when Surgical Registrar. It illustrates the association of liver abscess with malaria, and the tendency to recurrent attacks, commencing with rigors.

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The patient, a man, æt. 31 years, had lived abroad for nine years: in India for fifteen months; in Africa—south, east, and central parts—for eight years. He had always been strong and healthy before going abroad. When in India, the patient had an attack of ague, and suffered, on and off, from malaria the whole time when in Africa, which he left about two years before admission, in the interval spending ten months in Portugal, where, about a year before admission, he had definite syphilis.

Nine and a half weeks before admission, the patient felt pain in the right side, which, with loss of appetite, gradually became worse. Ten days later, the patient took to bed, but the pain continued to increase for about another fortnight, when an abscess of the liver was diagnosed, and three-quarters of a pint of greenish-yellow pus were drawn off. A drainage tube was left in for three weeks, a good deal of discharge coming away during the first two weeks; then, after three days' time, it almost ceased. The patient then felt very much better, and was able to get about again.

Nine days before admission, after having been up for a week, the patient had an attack of shivering, and a rigor, lasting three-quarters of an hour. During the night he was delirious, and in the morning he felt just as bad as before. He then slightly improved.

He was admitted to the hospital with a temperature of 102·2° F. The patient was fairly well nourished, but very anæmic. The right side of the abdomen and lower half of the chest were distinctly distended, and the epigastric and both hypochondriac regions were more prominent than natural. The upper border of the liver dulness on percussion was found to be at the levels of the sixth, seventh, and eighth spaces in the nipple-, mid-, and posterior-axillary lines respectively. The lower limit of the liver was felt (and percussed out) to be at the level of the umbilicus, in the right nipple line; and two fingers' breadths above the umbilicus in the middle line; the lower borders of the right and left lobes gradually passing upwards to the costal margins from the levels just indicated. In the right flank was seen the mark of the trocar used for tapping the liver, which was everywhere tender, but more so at the mark than elsewhere. The spleen was felt to extend vertically from the left costal margin to the level of the left anterior superior iliac spine. It occupied the left flank as far forward as the level of a vertical line drawn midway between the nipple and the anterior axillary fold. The notch of the spleen could be plainly felt half way down the border of the swelling in the left flank.

Chest, heart and lungs.—Normal.

The *blood* showed the red corpuscles unaltered in shape, none nucleated. There was a very slight increase in white corpuscles, the great majority being of the polymorphonuclear variety, a few lymphocytes with large single nuclei being visible. Only a very few eosinophile corpuscles were visible in the entire field ($\frac{3}{4}$ in. square). The condition of the blood appeared, therefore, to be one of secondary anæmia.

Temperature.—From the charts for some time prior to admission it was evident that a good deal of pus was still pent up, the temperature having been on five occasions during the previous three weeks up to, or over, 103° F., coming down to normal in the intervals. It was 101° F. the day before admission. Two days later it was 102° F., coming down to normal at 3 p.m., ascending to 102·2° F. at 9 p.m. the following day; pulse-rate, 84; respirations, 24 per minute.

Treatment.—The abscess in the liver was aid open by an incision in the right flank, passing downwards and forwards in the line of the exploratory puncture. A very large quantity of pus was evacuated, and the wound drained.

On the following day the temperature ran up to 103° F., but did not again go above 100° F. until the day before discharge from the hospital, thirteen days after the operation, when the patient was sent out with a tube still in, the discharge having greatly diminished.

Subsequent note.—A month after the patient left the hospital the tube was omitted; the wound then closed, the temperature having come down to normal with an associated gain in weight.

Case 3.—For the notes of the following case I am indebted to a valuable record* of eight cases of hepatic abscess by my friend, Dr. D. F. Dymott, Surgeon-Major I.M.S., Madras (retired). In this case the left lobe was involved. The excellent

* 'Trans. South Indian Branch Brit. Med. Assoc., March, 1885, Madras.

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notes and graphic description of the post-mortem appearances seem worthy of permanent record.

The patient was a very intemperate Hindu, anæmic, æt. 30 years, admitted October 1st, 1884.

History.—Fever for the last two months, with cough and severe pain in the chest. Expectoration abundant, and occasionally bloody. Twenty days ago his abdomen and feet began to swell.

State on admission. — There is considerable œdema of legs and feet. Abdomen much distended and contains fluid. No jaundice. No vomiting at any time. Temperature, 103·4° F. Expectoration consists of muco-pus and blood. Tongue covered with yellowish-white fur. Appetite bad. Bowels loose, the motions consisting of undigested food. Severe pain and tenderness over left side of chest. Below the second rib, the left side of the chest is absolutely dull on percussion. In front, breath sounds are only audible at the apex. Down the left back, there is dulness, the breath sounds being weak and accompanied by moist râles. The heart's apex beat is in the fourth left interspace, in the mammary line. Pericardial friction distinctly heard. No enlargement of the liver on the right side can be detected.

On October 7th, the abdomen being less distended, the lower edge of the liver was discovered about one inch above the umbilicus in the middle line. Some œdema was noticed over the left hypochondrium. Pericardial friction no longer audible.

On October 8th a large aspirator needle was inserted just to the left of the ensiform cartilage, and pushed backwards and inwards. Having struck pus, an incision was made with a long thin blade and twelve ounces of pus let out. Some of the pus was greyish, some reddish-brown. A probe passed 5½ in. into the abscess cavity. A large drainage tube was inserted. After tapping, the cough ceased, and the patient improved in every way for a day or two. He then got worse, the discharge became fœtid and scanty, and on October 13th he died.

Post-mortem.—A huge abscess cavity was found, part of the wall of which was gangrenous. The abscess opened freely into the left pleural cavity, and communicated also with a cavity in the substance of the left lung. The left lobe of the liver had almost disappeared, the thin layer which remained forming the floor of the abscess. The rest of the liver was uniformly enlarged, and studded everywhere with abscesses varying in size from a millet seed to a walnut. There was no ulceration of the intestines. The pericardium was thickened and adherent, except to a slight extent behind.

"The special point about this case was the difficulty of diagnosis. On admission there was no symptom strongly suggestive of liver disease. Only on the seventh day after admission was the enlargement of the liver detected. This, in connection with the other symptoms, and especially with displacement of the heart's apex *upwards*, made the diagnosis fairly certain. Here, as it does in so many other cases of disease, the position of the heart's apex beat afforded most valuable information."

Case 4.—The last case I shall venture to bring before your notice was under my care, and illustrates the condition resulting from at least two abscesses, one in the right extremity of the right lobe of the liver, pointing downwards; rupture through the lung occurred. The associated appendicitis and syphilis constitute a remarkable combination from which the patient, happily, recovered, after appendicectomy and drainage of the liver abscesses.*

A case of liver abscess, associated with appendicitis and gummata of the liver came under my care in October, 1904. A man, æt. 35 years, had been trekking in fever districts for two years in Portuguese East Africa and Rhodesia, and was subject to frequent attacks of malaria and "dysentery." For a month previously, on and off, there had been vomiting, and, for three days, constipation.

The temperature was 99·8° F., the pulse 128 per minute, the respirations 52 per minute. The man presented a most unhealthy-looking, cadaverous aspect, with clay-coloured, anæmic, wasted cheeks, and dark rings under the sunken eyes. The tongue was moist, and coated with a dirty white fur. The pain in the abdomen was referred to McBurney's point, and was equally severe in the corresponding position in the left iliac region. No swelling, or unusual resistance, could be detected

* For more complete report see 'Trans. Med. Soc. Lond.,' 1905–6.

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anywhere. No dulness in the flanks, or anywhere, in the abdomen, and there was no evidence of an enlarged or tender liver or spleen. From the first the man looked much worse than could be accounted for by the physical signs. Only on the fourth day was the pain localised to the right flank.

Two days later appendicectomy was performed, and a retrocæcal abscess, extending upwards beyond the reach of the finger, evacuated. The pus was offensive, dirty grey, and mingled with blood. The next morning anchovy paste purulent discharge escaped in large quantities from the wound, putting one on the *qui vive* for a hepatic abscess.

Tenderness over the liver occurred for the first time on the twelfth day after appendicectomy, and in the axillary line, over the ninth and tenth right intercostal spaces, and pus was drawn off at the second puncture with an exploring needle entered through the tenth space.

The liver was drained on the next day, the pus containing numbers of amœbæ. On the forty-sixth and forty-seventh days after draining the liver the occurrence of hæmoptysis suggested further exploration, and led to the discovery of a second focus of suppuration, about an inch and a half deeper in than the first. The second hæmoptysis was anticipated as the result of observations made on the pulse five minutes previously, and prophylaxis attempted at the moment of its occurrence. The two attacks of hæmoptysis had been preceded for five days by the presence of liver pus in the sputum.

The discovery of a deeper focus of suppuration on the day following the second hæmoptysis, apparently by relieving the tension between the second liver abscess and the lung, seems to have been responsible for the non-occurrence of hæmoptysis at any time subsequently. However, on the ninth day after the second liver abscess was reached the anchovy paste discharge from the wound became so profuse as to require dressing thrice daily, the prognosis seeming to become almost hopeless.

The excessive discharge at length yielded to iodide of potassium, pushed to gr. xxxv., four times a day, this large dose being tolerated for twenty-nine days before iodism occurred. The success of the iodide was subsequently proved to be due to the presence of a syphilitic taint.

The rapid return to the weight averaged six months before the present severe illness was noticed by the patient to be specially obvious when the profuse discharge was checked. This was very noteworthy, and was doubtless due not so much to the diminution of the discharge as to the effects of the large doses of iodide, which not only produced that diminution, but must have materially improved the general nutrition. As regards profuse night sweats, these occurred for the first time on the night of the appendicectomy, and continued more or less profuse for forty-one days (from the drainage of the liver for twenty-eight days). They ceased when severe hæmoptysis led to further exploration and the evacuation of a deeper focus of suppuration.

The patient left the hospital on April 12th, 1905, completely restored to health, with the appendicectomy and liver abscess wounds soundly healed. He remained in excellent health when seen on July 31st, 1905, and June 20th, 1906.

In this case, although the liver abscesses could not be definitely localised until twelve days later, owing to the complete absence of tenderness over the liver, and any other symptom or sign pointing to that organ, they seem clearly to have been in existence at the time of the appendicectomy. The patient stated positively that on no occasion had he ever passed blood or mucus during the attacks of so-called "dysentery" and fever, from which he had suffered for two years prior to admission to the hospital; and in the somewhat severe attacks of diarrhœa which occurred during the patient's stay in the hospital there was no blood or mucus evident. The presence of amœbæ in the liver pus seems to indicate that the abscess was primarily due to dysentery, and that the appendix became inflamed after the occurrence, and as a direct consequence, of the hepatic suppuration. Such a view would have its analogy in the variety of appendicitis complicating suppurative cholecystitis, a condition of the appendix which Dieulafoy considered secondary to that of the gall-bladder (Rolleston).

November 4th, 1907.

We have received from Burroughs Wellcome & Co. a copy of a little pamphlet entitled 'Anæsthetics, Ancient and Modern.' This is an historical sketch of anæsthesia that is really interesting from cover to cover. Will the author, in the next edition, tell us something about the fascinating block labelled "Hypnos" on the back of this delightful booklet?

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THE CLINICAL JOURNAL,

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EDITED BY L. ELIOT CREASY.

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A CLINICAL LECTURE

ON

GENITO-URINARY INFECTION, ESPECIALLY COLON BACILLUS CYSTITIS.

Delivered at St. Bartholomew's Hospital.

By C. B. LOCKWOOD, F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—I said when last we met that I would speak to-day on the subject of genito-urinary infection. I have chosen this because genito-urinary infection is often overlooked and is most difficult to treat. I cannot in the time speak of genito-urinary infection in general, and will, therefore, take one particular kind, namely, colon bacillus infection. I am about to speak of that because it is exceedingly common, easily overlooked, and easily mistaken for other conditions. It ought not to be difficult for you to understand that a patient who is suddenly seized with a rigor and high temperature, intense abdominal pain, perhaps, in the lower part of the abdomen, and who, at the same time, has increased frequency and pain on micturition, might be thought to be suffering from an attack of peritonitis due to inflammation of the appendix. Also that the frequency and pain on micturition were caused by pelvic inflammation caused by the appendicitis. Under such circumstances as those colon bacillus infection might, and often is, overlooked, and the treatment directed to some entirely wrong end.

I shall first speak of acute colon bacillus infection of the bladder, and have already indicated to you the mode of onset. The onset is often sudden and violent. The temperature may run up quickly to 103° or 105° F., the pulse may be accelerated until it is over 100. The patient is obviously in a state of high fever, and may have a rigor. At the same time he complains of most excruciating pain in the region of the bladder and especially about

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the neck of the bladder. The pain is said to be worst when the bladder empties itself, which it does very frequently, perhaps every twenty minutes or half an hour. At all events increased frequency of micturition is one of the most obvious and distressing symptoms. With such signs as those you would naturally proceed to make an examination of the patient, and, I think, under such circumstances your attention would be directed more particularly to the viscera which fill the lower part of the abdomen. The lower part of the abdomen in a case such as this will have assumed protective rigidity. You have often heard me say that when any abdominal organ is acutely inflamed the muscles become rigid to protect it from pressure. This is so not only as regards the urinary bladder, the gall-bladder, or the kidneys, but as regards any abdominal organ. More than that, the lower part of the abdomen over the region of the bladder is sure to be tender on pressure, and I have always observed that pressure is least well borne at the semilunar lines. There, of course, the abdominal walls are rather thinner, and it is probable that the pressure of the finger can be more directly thrust upon the bladder.

Besides the pain, tenderness, and rigidity in the front part of the abdomen, if you proceed to the next essential step and make a rectal examination, you will then make further discoveries. The finger having been introduced into the rectum the ordinary routine is to discover by pressure whether the prostatic urethra is tender or not—it is sure to be tender. Next the lobes of the prostate are examined, and will be swollen and exceedingly tender. After examining each lobe of the prostate in turn the finger ought to be directed to the trigone of the bladder, and the pressure upon it is almost certain to cause considerable pain. I have sometimes observed pain and tenderness in the situation of the vesiculæ seminales. It is easy to understand that an infective disease beginning in the bladder may spread along the ducts which communicate with the interior of the bladder or urethra. So that in colon bacillus cystitis one not infrequently observes that not only are the vesiculæ seminales swollen and tender, but that the inflammation has spread along the course of the spermatic cord, and that the testicle and epididymis have become tender. Every now and then the patient has a very severe form of orchitis.

I make this remark now concerning the abdominal symptoms. You may have observed that I have spoken of considerable tenderness in the lower part of the abdomen, and have said that the lower part of the abdomen has assumed protective rigidity. Clearly those signs indicate the presence of some tender organ, and the tender organ might or might not be the bladder. Such symptoms would correspond fairly well with an attack of peritonitis due to an inflamed appendix in the pelvis. That question will almost certainly arise. The resemblance to an attack of pelvic peritonitis, caused by some such inflamed organ as the appendix, is very close, because the patients will, in a serious and acute degree of colon bacillus cystitis, suffer considerably from abdominal distension and flatulence. I suppose the acute inflammation of the interior of the bladder affects not only the mucous membrane, but also the muscular wall and the peritoneum over it, and possibly the intestines which are in contact with the peritoneum. So that it is most desirable and necessary to search the pelvis carefully with a view of not overlooking the possibility of having really to deal with an attack of appendicitis. Occasionally I have known this confusion occur, and it has been supposed that the appendix has been lying on the back of the bladder, that the inflammation of the appendix has spread to the bladder, and then that the inflamed and suppurating appendix had actually perforated into the bladder, so causing colon bacillus infection and pus in the urine. However, I may say, as a matter of experience, that it is exceedingly rare for an inflamed and suppurating appendix, or an appendix abscess, to perforate into the bladder.

Supposing you have got thus far with the clinical signs of this acute and dangerous disease, the next step would be an investigation of the patient's urine. As regards the physical examination of the urine pathologists in their reports upon urine which is sent them say that it has some kind of odour. I observe that Dr. Andrews occasionally says it has a fish-like odour. I confess that odours do not appeal very much to my mind as clinical evidence, and question whether any conclusion is to be drawn from the odour of urine. I mention this because you might suppose that the colon bacillus produced in the urine the fæcal odour which it helps to produce in the contents of the alimentary canal. But this is not so; it is not safe to draw

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any conclusion from the presence or absence of odour.

The only other point about the physical characters of the urine which I think would attract your attention would be, first of all, that after standing it may deposit pus, or muco-pus. The presence or absence of pus in quantities would not go for very much. Sometimes the pus is so small in quantity that it is only discovered on microscopical examination. What, I think, is more constantly seen with the naked eye in colon bacillus urine are minute flakes of lymph and very often shreds, which on microscopical examination prove to be leucocytes, columnar epithelium, and colon bacilli, and which come from the prostatic ducts. A microscopical examination of the urine will always show that it contains pus also in great abundance, epithelium, and nearly always blood. Usually vast quantities of small, motile, oval-ended colon bacilli can be seen throughout the field of the microscope. I am speaking of course of acute cases.

When you have got that far with the examination of the urine the next step would be to have a proper bacteriological examination made, and you would probably have that made by an expert. An expert ought to be able to give you some indication as to the quantity of bacteria in the urine, because during the progress of a case a diminution of the number of bacteria would be an indication of recovery. And at last, when he is able to report that the infection had entirely ceased, you would begin to consider the patient as on the way towards recovery, if not well.

I have already said that it is of extreme importance to diagnose this dangerous form of genito-urinary infection. Unless you diagnose it, and unless you treat it properly, very dangerous complications may arise. Perhaps one of the most dangerous complications is the spread of the infection along the course of the ureters to the pelvis of the kidney, a condition known as ascending pyelonephritis. It is at this stage of the disorder that the surgeon is not infrequently brought upon the scene, because when you consider the train of the clinical effects which are associated with this ascending pyelonephritis you will agree with me that they simulate rather closely the formation of a stone in the kidney. It is easy to suppose that a patient with an enlarged kidney which is tender, and which, perhaps, has over it protective rigidity of muscles, pain in the course of the ureters, and blood in the urine—is easily supposed to be suffering from stone in the kidney. It may be difficult to disprove that belief. But usually on the clinical and photographic evidence the presence or absence of stone in the kidney can usually be decided.

Another, not perhaps dangerous, but very painful and severe complication of colon bacillus cystitis is inflammation of the prostate. I do not think I have ever seen a man with even a moderate degree of colon bacillus cystitis who had not a tender, swollen, and inflamed prostate. However, I have not observed that this form of inflammation of the prostate proceeds to the formation of abscess. But if the inflammation is allowed to go on indefinitely I agree with Mr. Herring and Mr. Daniel that it causes some forms of enlargement of the prostate gland.

I have not said anything to you about a further step in the examination of these cases of ascending pyelonephritis. But the endoscope and separator are often an assistance in diagnosis. It is not likely during the acute stage that the endoscope will be used. I have on various occasions looked into an acutely inflamed bladder, and should compare the appearance seen through the endoscope to an acutely inflamed optic disc and retina. But in the more chronic cases one only sees some small specks of lymph waving and floating about over the inner surface of the bladder. There is considerable dilatation and engorgement of the vessels of the mucous membrane, and usually this is best seen at the ureteral orifices. The papillæ at either of those orifices may be swollen and their mucous membrane inflamed, and this would cause one to suspect ascending pyelonephritis. Sometimes the mucous membrane lining the prostatic ring is inflamed, swollen, and easily bleeds with the slightest movement of the endoscope. That, of course, helps to explain the origin of the blood found in the urine.

When pyelonephritis has occurred it is of extreme importance to determine what has taken place in the affected kidneys. In one such case where I was called in to remove a suppurating left kidney, we found, by using Luy's separator, that the suppuration in the left kidney had ceased and that from that side a little clear water was flowing. But urine full of pus and the colon bacilli was

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flowing from the right kidney; so that obviously, an operation on the left would have been useless.

Colon bacillus cystitis sometimes occurs after an injury to the genito-urinary tract. A little while ago a gentleman sustained a severe accident, and the evidence went to show that his right kidney was seriously lacerated. Perhaps other organs were injured at the same time. He recovered from this injury but never regained his strength. After a very acute attack of cystitis, which I have no doubt whatever was an attack of colon bacillus cystitis, he was left with a bladder which was irritable—it acted two or three times during the night—10 per cent. of pus in the urine, with blood, and an infection which, according to the bacteriological report, was colon bacillus. He had, in addition, a very common complication of colon bacillus infection, namely, *Bacillus proteus vulgaris.* This caused a slight difference in the urine. I ought to have mentioned that the colon bacillus is found in acid urine. Throughout the whole of the illness the urine may remain perfectly acid. If the urine becomes alkaline—it was once very alkaline in this particular case—then the probabilities are that some form of proteus vulgaris is there in addition. This gentleman may have had the colon bacillus and proteus vulgaris introduced by catheterisation. He had one curious symptom and for which I saw him. He had on the right side a total absence of pain and even of tenderness, but on the left he was always complaining of pain. This had incapacitated him from following his business. It had been assumed that his lacerated right kidney had developed a stone, for the skiagram showed a shadow, and that the pain on the left side was a referred pain, which is not unusual. I am very sceptical about referred pains, so that we proceeded as follows: First of all his bladder was examined with the endoscope. I have just described to you the appearances seen with that instrument. The right ureter—the side of the lacerated kidney—had a minute, pale orifice, and I saw no urine come out of it. The left ureteral orifice was swollen and red, and I saw urine and flakes of pus coming out. The bladder was then washed out and a separator introduced. From the right side a minute quantity of very thin urine was obtained and from the left side a large quantity. The bacteriological report says that the small quantity of urine from the right side contained no bacteria, but that from the left contained pus-cells and an abundant growth of colon bacillus and of *Bacillus proteus vulgaris.* What is the meaning of that? On the right side the infection had subsided, but on the left he had still proteus and colon bacillus infection of the ureter and pelvis of the kidney, and which had ascended from the bladder. For this he is undergoing a lengthened course of treatment by urotropine and intravesical injections of nitrate of silver, and with considerable benefit.

I will now mention two cases of colon bacillus cystitis to show you the ease with which this dangerous and troublesome affection may be overlooked. A gentleman had a small operation performed. A hydrocele of the left side had been treated by the radical operation. He made a very rapid recovery, but about three weeks afterwards came with acute inflammation of the right testicle. How natural to think that an operation such as that should be followed by an acute inflammation, say, of the opposite organ. But being of a sceptical frame of mind I proceeded to make a few inquiries. First of all he told me, when questioned, that he had had greatly increased frequency of micturition since the testicle had been inflamed, and had had to get up many times in the night to empty his bladder, and, in addition, the act of micturition was exceedingly painful. I then made him pass water in my presence. You should always make these patients pass water into a glass in your presence. In that way you will often make discoveries. He passed urine which was turbid and contained numerous flakes of lymph and prostatic threads. On inquiring as to what had been happening during the last week or ten days he informed me that he had had a violent attack of influenza. Whether he had had influenza or not I do not know, but the bacteriological examination of that urine showed that he had colon bacillus cystitis. How easy it would have been to have overlooked the infection of the bladder, and to have attributed the increased frequency and pain on micturition to the orchitis.

Here is another case in which the diagnosis was rather difficult. A patient was brought by an old friend of mine (Dr. E. S. Tait) because he had recently begun to pass water with great frequency. The act of passing water had been attended with pain, but not excessive pain. Otherwise the patient felt well and there seemed hardly anything to explain

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his symptoms. I need hardly go over the clinical examination, for he had symptoms such as I have already mentioned—pain and tenderness over the front of the bladder, and *per rectum* marked tenderness at the neck and trigone of the bladder and of the prostate. If a patient has anything tender in the genito-urinary tract, the prostate and the trigone are the parts of the bladder which you can get at best with your finger, and there is no protective muscular rigidity to hinder you from making a proper examination. This man was requested to pass water in my presence. He passed a small quantity of perfectly clear, natural urine. Judging from the prostatic tenderness and the tenderness of the trigone of the bladder and so forth, I guessed that he had some infective disease of his bladder and prostatic urethra. In a little while he passed some more urine full of flakes of lymph and pus. Doubtless the pressure upon the prostate and neck of the bladder had squeezed these out. The bacteriologist who examined the specimen of urine reported that it was teeming with the colon bacillus. How easily this might have been overlooked.

Now how does this dangerous colon bacillus get into the human body? I cannot go over all the points, but am struck with the fact that many patients who have had a prolonged and dangerous form of colon bacillus cystitis have had some antecedent illness, and one of the commonest, perhaps, was a severe attack of influenza. But it is so customary nowadays to call every feverish affection influenza that one cannot help feeling sceptical on that point, and one would like to have more scientific evidence. But whatever may be said about an antecedent cause, there is no question but that the colon bacillus descends from the kidneys. I have no time to go into all the evidence which shows that bacteria in the general circulation can pass through the human kidneys to the bladder, but there is abundant evidence to prove that pyogenic bacteria and intestinal bacteria, more especially the typhoid bacillus, can pass through the kidneys and descend the ureters and affect the bladder. Why they should implant themselves on one bladder and not upon another I cannot say. But some of these cases of severe colon bacillus cystitis occur in men who have some obstruction to the exit of urine—stricture or some enlargement of the prostate—which prevents them entirely emptying their bladder. It not infrequently occurs in females who cannot empty the bladder. For instance, the last case of colon bacillus infection that I saw was in a lady whose bladder held a large quantity of lotion when it was washed out—it easily held a pint. And whenever the lotion was put into the bladder we had great difficulty in getting it out again. Her bladder was atonic, it refused to contract.

Colon bacillus cystitis is not rare after rectal operations. It may have entered the bladder *per urethram;* but it is quite possible that the bacillus may pass into the circulation from the wound and be carried onwards through the kidneys into the bladder.

I had intended to say something on the subject of chronic colon bacillus cystitis. When once the colon bacillus enters the bladder it may lurk in it, in the prostatic ducts, or in the vesiculæ seminales, for an indefinite period. I have under my care a gentleman who had a rectal operation performed five years ago. He had an instrument passed for retention and then had a violent attack of cystitis. Strenuous efforts had been made to cure this. At last the frequency of micturition became a great trouble. He could not eat his dinner without having to pass water, nor play a game of billiards. His urine was reported by Dr. Andrewes to contain strings of columnar epithelium mixed with bacteria, which were proved by culture to be colon bacillus. I need not say that the clinical symptoms fully bore out these bacteriological details. I cannot help thinking that this gentleman had had colon bacillus infection of his prostate for at least five years. If I had time I could produce evidence that patients may go on with colon bacillus infection of the bladder, and the ureters, and the pelvis of the kidneys for many years. If I had known as much about colon bacillus infection of the bladder, ureters and pelvis of the kidney five years ago as now I should never have explored the right kidney of a girl to see whether it contained a stone. That patient still has colon bacillus infection of her bladder, but is slowly getting better after washing out with nitrate of silver, and after vaccination with colon bacillus.

As regards the treatment. In acute colon bacillus cystitis the patients are exceedingly ill and are, of course, in bed. They have to be carefully nursed and require suitable diet. This severe form of cystitis may go on for many months.

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The acute stage may last one to three months, but the chronic stage many years unless properly handled.

As regards the treatment by drugs, urotropine, helmitol, hetraline, and such like are of use. Perseverance is the most essential thing about the treatment by drugs. It requires a long course of treatment by these internal antiseptics before you will produce any result. From ten to fifteen grains of one of them three times a day in a quantity of fluid is probably best. I myself have never seen any harm result from their prolonged use. Sandal oil is sometimes useful. It tends to relieve the pain at the neck of the bladder during the act of micturition. I have never known sandal oil to do any more harm than upset the digestion.

It is better not to wash out the bladder during the acute stage of colon bacillus cystitis; but during the chronic stage it is one of the most valuable adjuncts.

It will be necessary for you all to know with perfect clearness how this very important little operation in surgery should be carried out. Unless it be carried out properly the patient will from time to time have fresh forms of infection introduced into the bladder, and you will not kill the bacteria. Mr. Marshall, of Messrs. Croydon & Co., has very kindly sent the apparatus which is used for washing out and disinfecting the bladder. First of all I need hardly say that you must bring to your aid all the resources of aseptic surgery. The hands must be disinfected as carefully as they are for opening a knee-joint. The opening of the urethra must be disinfected to the very best of your ability. All the instruments should be boiled or very carefully disinfected with efficient antiseptics. Boiling is the safest and best way. These are the articles which you generally find convenient to use: First of all here is a suitable syringe to put in 5 per cent. solution of cocaine. Patients are very anxious about introduction of anything into the urethra. It is reasonable, therefore, that they should not be subjected to pain, and that they should have all the confidence which a little cocaine gives them. Next the antiseptic is placed in a glass reservoir with water and the catheters are soaked in that. As a matter of fact the catheters ought to be placed in that reservoir at least half an hour before the washing out is done. The catheters from time to time ought to be boiled. After the catheter has been disinfected it is taken out of the disinfecting solution with disinfected hands and some kind of lubricant is placed upon it. I generally use glycerine. I am fully aware that glycerine is objectionable; it smarts a little, and makes the urethra dry. But if vaseline be used the wall of the urethra is coated with it, and bacteria in the urethral follicles, or on the mucous membrane, are covered over so that the disinfectant cannot get at them. The catheter having been passed the next step is to empty the bladder. Perhaps the patient has emptied it, but do not trust to that, but wash the bladder out with boracic lotion to get rid of the last of the urine. The next step is to introduce four ounces of a solution of nitrate of silver in distilled water. If any urine had been left in the bladder it would neutralise the nitrate of silver and render it inert. Of all the antiseptics which are introduced into the human bladder I should say that nitrate of silver and distilled water is by far the best and most efficient. Begin with $\frac{1}{10}$ gr. to 1 oz., and gradually increase the strength until it causes slight pain or discomfort. Nitrate of silver is not the strongest germicide, but you have also to consider the treatment of the mucous membrane of the wall of the bladder. Healthy mucous membrane of any organ, whether of the urinary bladder, the gall-bladder, the stomach or elsewhere, has extraordinary powers of resisting bacteria, and if you can, by the judicious use of nitrate of silver, separate the lymph and bacteria from the surface of the bladder and make its epithelium healthy again, you have gone a long way towards curing the infection.

There is one last point about treatment which I will just mention. We are living in times of change, and when you are called in to treat a case of colon bacillus cystitis you may be questioned as to the propriety of vaccinating the patient. We have tried vaccination with the colon bacillus and we are watching the result. But it is difficult to satisfy yourself from mere clinical evidence as to the efficiency of any particular method of treatment. Nevertheless vaccination is a right and proper thing to do, and I trust that at some future date we may be able to report to you that we have had successful results from it. Certainly we do think that from this method of treatment we have obtained better results in some of our other forms of infection, especially infection with the *Staphylococcus pyogenes aureus*. Let me just add that every man in practice ought to thoroughly understand the apparatus for washing out the bladder and how it is used.

November 11th, 1907.

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A LECTURE

ON

BERI-BERI.

Delivered at St. Thomas's Hospital.

By F. M. SANDWITH, M.D., F.R.C.P.,
Lecturer on Tropical Diseases at the Hospital; Physician to Out-patients at the Seamen's Hospital, and Lecturer at the London School of Tropical Medicine.

GENTLEMEN,—There are many names given to the disease which we call "beri-beri," but I do not think it is necessary to bother you with them as they are not of any importance in this country. And, of course, calling a disease by a variety of names does not help us to understand it or to find out its cause. But one name by which it is known is "kakké," which means a disease of the legs in Japan. Some of you heard Baron Takaki's lectures on beri-beri in the Board Room of this hospital a year ago. He showed us some very good diagrams, and told us definitely how he and others had succeeded in stamping out beri-beri from the Japanese navy. He believes that beri-beri used to affect almost every individual in the Japanese navy before that time, that beri-beri was caused by an insufficiency of food and an insufficiency of nitrogen in the diet. Whether his view is correct or not we can only say that he has done most wonderful things in reducing both the sick rate and the mortality in the Japanese navy. A definition of the disease, if you want one, is this: a specific multiple, peripheral neuritis, of many tropical and sub-tropical climates, which occurs both epidemically and endemically. It is an infectious disease, of unknown cause; it may also appear as an epidemic under certain conditions in temperate countries, such as Ireland. When I am talking about peripheral neuritis you will be thinking of those cases of peripheral neuritis which you have seen, and I am assuming to-day that you have never seen beri-beri. You have seen neuritis occurring after arsenic, and after lead poisoning and alcohol and diphtheria. Peripheral neuritis does not vary very much in its symptoms although the causes vary tremendously. With regard to history, I can tell you that there are records of this disease in China, where it is also called "kakké," since the second century before Christ, so it has been known to the world for at least two thousand years. The Dutch know about it, and the literature concerning beri-beri is tremendous in extent, as it is about most of those diseases of which we do not yet know the cause. I will not mention the whole of the geographical distribution, but there are three important endemic centres in the world. One of these is the Dutch possessions in Southern Asia, another is Japan, and the third Brazil. Those are the places where you would go to-day if you wished to study beri-beri. You would be certain to find it there because it is endemic. There are many other places, such as Sumatra, India, Burmah, Siam, Singapore, China, Korea, Japan, Torres Straits. Also it is found in Morocco, St. Helena, Madagascar, Gambia, Australia, New Fiji, South America, Guiana, besides Trinidad and Jamaica. It is a disease not only existing in the various localities I have given you, but more or less over the tropics, but it is the imported cases which concern us in this country. There is, apparently, no limit to the distance over which beri-beri can be transported by ships, and it is chiefly by sea carriage that this disease has been and is carried over the world. You know that in regard to other infectious diseases, smallpox for instance, you will find in books in which the subject is discussed that it is safe to build a smallpox hospital at a certain distance from a town, but not in the middle of the town, that there is a distance beyond which it is safe to build such a hospital because the disease will not be spread. But it is a different matter with beri-beri; it may be transmitted from India or from China to the London Docks. The disease develops very often in the crews of ships coming from those parts, those crews being Asiatics, and it may develop months after the ship left the infected port, and even months after a ship has been lying in an uninfected river. In most of those instances the disease remains confined to the ship on which it broke out. That is to say there is a focus of infection on the ship, and it does not seem to be strong enough to spread to the new country. In ship epidemics, dirty, crowded, and ill-ventilated vessels seem to supply the necessary factors for the outbreak and persistence of beri-beri if the crew are given improper food. I have mentioned already some of the predisposing factors, and it is interesting to note that formerly a quarter of the navy of Japan was annually attacked. On

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the ordinary steamers used by passengers nowadays beri-beri is found only to attack coloured people. However, many cases, especially in northern latitudes, have been found on sailing ships which have been called beri-beri, but which are probably not instances of that disease. Dr. Nocht, the superintendent of the Hamburg Tropical School, showed some years ago, and his views have been accepted by others, that many cases of what was thought to be beri-beri on sailing ships were a form of scurvy with neuritis which simulated beri-beri. It has been assumed for many years that a neuritis on board ship must be beri-beri. But I venture to suggest that, at least in northern latitudes, it is not so. We see every year in London many cases of beri-beri, which have been brought to this country, cases of ordinary peripheral neuritis coming from the Malay States, India, Japan, or somewhere in the Far East. The disease which Dr. Nocht wrote about, pseudo beri-beri, is due to bad food, and affects the officers more than the crew, and he suggests that this is due to some mouldy flour or bread taking the place of the old ship's biscuits. He points out that although the ships must still carry biscuit, they carry a limited amount of flour to make into bread, and they naturally give such bread as a luxury to the officers of the ships and not to the men. Dr. Nocht maintains that it is a recently discovered disease and that it is now increasing. In 34 ships where it was discovered, 12 of them also had scurvy on board, and some others of the 34 had cases of bleeding gums, though there were no other symptoms of scurvy. I have only mentioned this to clear the ground, and we need not bother further about it, because it is not true beri-beri.

Real beri-beri occurs in epidemics, in institutions, and in gangs of men. It is one of the diseases which comes from over-crowding, and of course you know of others. It is found in schools, if they are over-crowded and ill ventilated; very often it is present in prisons; in Singapore, for instance, 124 prisoners were attacked in 1898, and in Madras there was another outbreak in the prisons that year. It is found in miners, for instance, in tin miners in Japan, in coolies on estates, and in railway labourers on the Belgian Congo. In the Japanese war during eighteen months of 1904–5 there were 97,572 new cases of beri-beri in the army. These are Baron Takaki's figures, and out of these 3956 died, which gives a mortality of four per cent. So the disease is by no means stamped out of the Japanese army, and Baron Takaki says it is because they will not accept his views as to diet. Now we have to see its connection with lunatic asylums. In 1896, in Alabama, U.S.A., there were 71 cases of beri-beri, in both whites and negroes, out of 1200 inmates, and 21 died of the disease. At the same time there was an outbreak in the State Asylum at Arkansas. Then, to come nearer home, at the Richmond Asylum, Dublin, between 1894 and 1897 there was beri-beri constantly present for three years, 535 cases, and 44 of them died. The number of inmates at that time varied between 1500 and 1800. The asylum was built for 1000, so you see these lunatics were overcrowded. Then again, although beri-beri was present during these three years, the new cases chiefly occurred during the months of July, August, and September, which of course are the months during which the climate of Dublin was doing its best to become temporarily tropical. During the same years there was an outbreak in the lunatic asylum at Melton in Suffolk, and there are records of cases in other asylums in Germany and France. In the Japanese navy beri-beri was almost eradicated by improving the rations of the sailors, adding meat to their diet, and barley to the rice, in the proportion of 3 of barley to 7 of rice. The Japanese found it was not good to be entirely cereal eaters, and that if meat was added to their food they were able to do more work and they suffered less from beri-beri. If it had not been for this discovery it would have been impossible for Japan to have conquered China in the war, and of course much less possible for her to have succeeded later in fighting Russia. In some divisions of the Japanese army beri-beri has been reduced by similar measures, by improving the rations and giving this mixture of barley with the rice. For instance, in the Osaka division of the army in 1883 in 1000 soldiers there were 308 cases of beri-beri, that is to say every third man was in hospital with it. In 1884 there were 232 cases out of 1000, but in 1885, when Baron Takaki had gone back from St. Thomas's Hospital and started this new ration, the number of cases was reduced to 7 in 1000. Those figures are very remarkable. I naturally quote the most striking, but they are all remarkable. That is in one division of the

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army only, and you see that the disease affected the Japanese navy and army similarly. We do not know the cause of beri-beri, as I have said, but that is all the more reason why we should discuss various points about it which may possibly have relation to the cause. There is some mystery, or what seems to be a mystery, lying under our noses which we have not yet found out. Not every man who lives in the east is a rice eater, but every beri-beric in the east is a rice eater. When we say "a rice eater" we mean a man who eats it regularly, and eats it like we eat bread as the staple cereal, that is to say from one to one and a half pounds of dry rice in the day. The converse of that is true, for people who do not eat rice do not get beri-beri. That is quite true of the east, and apparently true of every other part of the world. There are many different varieties of rice and many different ways of cooking it, but there are two particular ones which I shall tell you of. One is cured rice and the other is called uncured rice. Cured rice is the raw grain, soaked and then *boiled*, and then dried before it is taken to the mill to be winnowed in order to remove the husk. That is the preliminary stage in the preparation of rice before it goes to the cook. The uncured rice is not heated in water before it is milled, and Dr. Braddon, of Malay, states that beri-beri is due to the use of this uncured rice. He has not succeeded in converting everyone to his views, but he has written an interesting book on the subject. Beri-beri is an infectious disease chiefly occurring in low-lying parts of large towns, and it flourishes in damp heat. It is possible that a want of food, or the taking of improper food, may be the predisposing cause. But an entire absence of food, such as during a famine, is not enough to cause this disease, and it does not accompany or succeed famines. Healthy, perfectly sound rice cannot be the cause of the disease. In the Molucca Islands, East of Borneo, beri-beri exists, but the natives eat sago, not rice. The cause of the disease must evidently be a specific one, originating outside the body. Is it possible it can be some germ affecting the rice or the flour of rice and producing the chronic toxin after introduction into the human stomach? It is often present in different parts of the world, in those people who live chiefly on imported rice which has become mouldy. Those of you who heard last year's lectures on pellagra and maize will remember that there is some analogy between the spinal cord disease occurring as a result of the habitual use of damaged maize, and the suggestion thrown out by many people that there is something in the rice which poisons the individual—that is to say, that there is a special toxin connected with the rice. But that is not the same idea as a deficiency of nitrogen, which some of the Japanese so firmly believe in. Dr. Hamilton Wright, who has worked well at the subject, believes that all beri-beri is preceded by a stage of gastro-duodenitis. If that is so it is an additional reason for supposing that the poison enters by the mouth and sets up some irritation of the stomach and duodenum, which afterwards ends in a definite toxin and the production of neuritis analogous to post-diphtheritic neuritis. Many micro-organisms have been at different times suggested as the possible cause of beri-beri, but none of them are believed in. Le Dantec, the French writer on tropical diseases, boldly includes beri-beri under the heading of "Diseases of Food Origin," and he considers it comparable with scurvy. There is no evidence that the poison of beri-beri is conveyed by the earth or by the air, or water, or sewage. So many diseases have been found of recent years to be caused by some intervening parasite that here, too, it has been suggested that there may be some intermediate host, and Dr. Daniels has proposed that if there is an intermediate host it may be pediculi. But if we do not know what the poison is, it is difficult to look for it in lice. The disease is not contagious, but it is transmitted by human intercourse, and it may be transmitted by damaged food, such as rice, or by the clothes of patients who have suffered from the disease. Then there are also other predisposing causes which I could mention to you. For instance, people who live in houses with damp foundations are liable to be attacked by the disease, and coloured races are more likely to suffer than whites. There is also a different incidence in regard to sex, for women are less liable to be subjects of the disease than men, probably because they are more protected by their mode of living. With regard to age, sixteen to twenty-five years seems to be the period of greatest liability. Overcrowding, bad ventilation, and bad hygiene I have already mentioned. Any previous illness, especially if it be an acute one, will predispose, and two predisposing

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factors in women are pregnancy and confinement, and the subsequent suckling. Anything which weakens the individual makes him or her liable to get the disease, but that of course is not peculiar to this complaint. New-comers entering an endemic area are more liable to suffer than are the natives in that area. There is a short period of incubation, say seven to twenty days, and a period of exposure of less than three months is requisite for the full development of the disease. It is unscientific to talk about the different varieties of the disease, but it is exceedingly convenient from the clinical point of view. There are four *varieties* which are usually mentioned: (1) First of all there is the mild or sub-acute variety, with weakness of the legs, slight anæsthesia, and œdema over the tibia, palpitation over the heart, and pain on pressure on the calves, and anæmia. The mild attacks may last for months or even years, becoming worse in the summer and better in the winter. I want you to notice at once that the disease begins in the legs, a peripheral neuritis beginning from below and working upwards, and in that it differs from some other forms of peripheral neuritis, which begin with the wrist drop. (2) Next there is the dry or atrophic variety, with an onset which is gradual, beginning in the legs and followed by an affection of the arms, until the patient becomes bed-ridden from inability to use the muscles. The limbs of the patient are very sensitive to the touch; he emaciates, and is reduced to a condition such as you see in these pictures. Palpitation of the heart and œdema are usually not present in this form, as the poison seems to have spent itself chiefly on the nerves and muscles, leaving the heart comparatively free. But even here death may ensue from endocarditis or pleurisy. Recovery is very slow, extending over more than a year. (3) The third variety is the moist, dropsical or œdematous. This may develop from the dry form, but more often from the mild variety, that is to say from No. 1. Paralysis in this is not so marked as in the dry form, but the heart symptoms are much more evident. There is œdema of the legs and body, palpitation of the heart, effusions into the serous cavities, pressure in the epigastrium, and diminution in the urine, but the recovery is less slow than in the dry form. (4) The fourth and worst variety is called the acute, pernicious, or cardiac. This may be acute from the onset or it may suddenly become acute from a mild case. The patient may find himself bed-ridden after only a few days' illness on account of his paralysis or from the seriousness of his heart symptoms. In the Dutch Indies there are stories of soldiers having done their duty in the morning and dying of beri-beri on the evening of the same day. But it is fair to assume that in those cases there were previous mild symptoms which had not been recognised.

Now as to the symptoms of the disease. The typical beri-beri patient walks like a man emerging from the water with his wet clothes on, or somebody walking in stiff clay, for he cannot lift his legs from the ground. The common symptoms, besides weakness of the legs, are: slight œdema over the tibia, tenderness of the calf muscles and an increase in the knee-jerk, dilatation of the stomach, loss of appetite, tenderness over the epigastrium and a sense of fulness there. But gradually there is a weakening of the peroneal and anterior tibial muscles, and foot-drop, and other muscles may be affected, such as those of the arm and shoulder. In severe cases there may be paralysis of the diaphragm. Often there is irritation of the vagus, a rapid pulse going up from 80 to 140, palpitation of the heart, retching and vomiting. Hyperæsthesia of the skin is rare, but anæsthesia or altered sensation is common. The skin reflexes are normal, but the knee-jerk in an advanced case is either greatly diminished or entirely absent. This may be preceded, as I have said, by an increase of the knee-jerk at the very beginning of the illness, that is to say during the first few hours or days of the illness, which is probably before you will see the patient. The right ventricle of the heart is dilated and eventually becomes hypertrophied, the left heart becomes dilated afterwards. If the diaphragm is paralysed and raised the impulse of the heart may be in the fourth intercostal space outside the nipple line. The urine may be suppressed entirely, or diminished to only one or two ounces in the twenty-four hours, but in the dropsical form a patient who is convalescing may pass from four to seven pints of urine per day, and with the polyuria the œdema will disappear. The urine contains neither albumin nor sugar. The greater the quantity of œdema the less the quantity of urine passed.

In the third or fourth week of the illness there is generally a subsidence of the symptoms, and as in other diseases of nerves the lesions which are last caused are the first to disappear. The epigastric symptoms improve first, and the œdema goes with the onset of polyuria. The knee-jerk may remain absent for as long as one and a half to two years. A neuron partially destroyed may recover with difficulty, but in all degenerations of nerves a totally destroyed neuron can never recover. The mortality from the disease at the present day is not excessive. In ordinary civil life in Japan the mortality was something like 3 per cent., and

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in the Dutch-Indian army the mortality was from 4 per cent. to 5 per cent. But in the old days the disease was much more dangerous. For instance, in 1873 in Brazil there was said to be a mortality from it of 50 per cent. to 74 per cent.; fortunately in all countries beri-beri is much less fatal than it used to be. The disease may last from ten to twenty years. In acute cases death usually takes place from paralysis of the heart, sometimes from embolism of the pulmonary arteries, and sometimes from uræmia. In chronic cases death is due to general exhaustion, or to the presence of some concurrent disease, such as tuberculosis, malaria or dysentery.

Post-mortem appearances will of course differ according to whether the case is acute or chronic. Let us first consider the post-mortem appearances of acute cases. There is early rigor mortis, and usually there are some signs of gastro-duodenitis, which I have told you may be the primary lesion. The mucous membrane of the stomach is congested and spotted with small hæmorrhages, and the intestine is affected in that way in some cases as far as the cæcum. Sometimes there is erosion and then sloughing of the necrosed mucous membrane. In these acute cases there is also bilateral symmetrical toxic change in the peripheral terminations of many or few sensori-motor nerves, such as the vagus, cardiac, pulmonary, laryngeal, and sometimes the phrenic nerve. Again, the nervous system of the heart is deeply implicated. The right heart, as I have already told you, is dilated, and there is a dissociation of muscle fibres and fatty degeneration. The amount of œdema varies, and the serous cavities will be found to contain clear straw-coloured fluid. At a post-mortem on an individual who has died after two or more years' illness you will find greater changes. There will be some muscular wasting confined to the legs, or possibly affecting also the arms and trunk. There is no gastro-duodenitis, the chief visceral lesion being cardiac. The right heart is dilated and hypertrophied, the left heart usually dilated but not hypertrophied, and there may be extreme fatty degeneration and brown atrophy of the heart. In the lungs there may be emphysema, hypostatic congestion of the bases, and occasionally thrombosis, the liver congested and fatty, the spleen increased in size. In the kidneys hyperæmia and cloudy swelling will be seen; the muscles are atrophied, the degree of atrophy depending on how far their nerves are degenerated. The nerves themselves show chronic degeneration, which is no longer strictly peripheral. In the posterior spinal ganglia and in the anterior horns of the cord are numerous cells in a condition of extreme degeneration. The cortical, cerebellar and cerebral cells are always normal. Hamilton Wright has reported a case of five years' standing with marked degeneration in the sacral and lumbar fibres of Goll's columns, a peripheral neuritis apparently ending in disease of the cord. I would remind you that ankylostomiasis, especially when associated with pellagra, which is a cerebro-spinal disease, may present appearances very like those fat, swollen youths which one sees in pictures of beri-beri. But there is no sensitiveness of calves on pressure, because they are not cases of peripheral neuritis. As a matter of fact you have first to make up your mind that there is peripheral neuritis, and then you will see you can exclude all the diseases which are not peripheral neuritis. Then you must consider what can have produced the disease. And if I tell you about two cases which occurred in the Egyptian army you will see that sometimes it is difficult. First of all one man, and later a second, was found to be ill, both recruits, coming from the same village. It was thought that they were both suffering from beri-beri, and I was asked to see them because I had stated that beri-beri did not exist in Egypt. The two individuals had been kept in the army hospital for several months, and it was not known that they had come into contact with any source of infection which could have made them ill. It was thought that as they had both come from the same village there might be some local infection in the village, and I got someone to go down there to investigate matters, to see if there were any other cases, but there were none. Some time afterwards, when people were still in doubt as to the diagnosis, a man confessed that he made it his business to supply arsenic in large doses to the recruits who wished to malinger. The conspiracy was discovered on account of his giving too large a dose of arsenic to one recruit, who as a consequence suffered from profuse diarrhœa. It then became clear that these two cases were instances of arsenical poisoning, and that they had been able to renew their symptoms by taking extra large doses of arsenic under the noses of the hospital authorities. Speaking generally, beri-beri is the most likely form of peripheral neuritis for you to meet with either among Orientals or on ships which have come from the East.

The prognosis in this disease is always doubtful, because of the heart. Even in a severe dropsical case there is quite a good prognosis if there are no heart symptoms. Vomiting is a bad sign in the disease. Signs of improvement are the onset of polyuria and the return of the appetite. I need hardly say that concurrent diseases always render prognosis unfavourable.

Now as to *treatment*. There is no specific remedy known for this disease, partly because we do not yet know the cause of it. There is no particular method of prevention known either. But so far as possible you would naturally try to remove patients from the place where they caught the disease, that is to say, from the house, or

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prison, or ship. There are many heart remedies which have been tried and which have been found useful, especially those which will act on the kidneys as well. Digitalis and other diuretics generally act so well that tapping of serous cavities is not necessary. You can see from these pictures that the men are sometimes waterlogged, but in spite of that in favourable cases the œdema disappears, the water being carried off by the kidneys. Strophanthus, caffein and diuretin, and camphor injections, which are not very often used in this country, are all useful. The camphor is used in 1 in 10 of sterilised oil. Nitro-glycerine is sometimes useful, and for the dyspepsia and vomiting you need to give hypodermic injections of strychnia and morphia. During convalescence the patient requires tonics, and chronic patients will want galvanic electricity, massage, and warm baths to help them to get well. One of the most important things to insist upon in all cases which come under your care is rest in bed, because you cannot be sure how the heart will be affected. You must keep the patient lying in bed until you find he is able to sit up in bed and eventually to move out of bed without his pulse-rate undergoing any great increase. It is a safe rule in the case of every beri-beric to put him to bed and keep him there under rigid precautions until you are certain his heart is not troubling him. There must be careful dieting, and a large quantity of milk is very good. Some orientals do not like milk, but you can often persuade them to drink it.

With regard to *prevention* I cannot tell you very much, except that there must be absolute purity of food and water, and see that the houses are well drained, with a damp-proof course to keep the moisture from rising from the soil into the house, and there must be good ventilation. In the case of infected houses the inhabitants must be evacuated and the whole place properly cleansed and disinfected, and it is also necessary to disinfect linen and clothes. Some of you may, in the future, go out as surgeons on steamers, and if you do you must be careful to be on the look-out for beri-beri when examining recruits for the service. Beri-beri is one of the first things to look out for under those circumstances. I have had figures supplied to me by the courtesy of the chief medical officer of the P. & O. Company. They employ 7000 natives on their ships, and the cases of beri-beri among them have been reduced from 44 in 1902 to 10 in 1905. And this is due entirely to the more careful examination of the native recruits in Bombay. For instance, only one case was brought home to London in a P. & O. boat in the year 1905, and that fact shows that the disease can be tremendously diminished by taking great care about the medical examination of recruits in beri-beri countries.

November 11th, 1907.

SOME UNUSUAL CASES OF DIABETES.

By J. R. BRADFORD, M.D., F.R.S.,

And Discussion* thereon by Dr. Pavy, Dr. S. Phillips, and Dr. Kingston Fowler.

The term "diabetes" probably includes a number of conditions dependent not only on various causes, but also associated in all probability with different lesions, and the view is very generally held that only a proportion of cases of diabetes can be associated with lesions of the pancreas. Still it would seem as if the number of cases of the malady that can be attributed to a pancreatic origin is increasing. There is still much difference of opinion as regards the nature of the pancreatic lesions associated with diabetes, although most observers have recorded atrophy and a peculiar variety of diffuse cirrhosis as the most common. Diabetes has been found in association with other pancreatic lesions, as, for instance, calculi, cystic disease, and new growths; but all these pancreatic lesions have frequently been described as occurring without diabetes. In some very rare instances glycosuria and diabetes have been found associated with acute lesions of the pancreas, but there is a general concensus of opinion that diabetes and glycosuria are but rarely seen in association with such acute lesions as acute hæmorrhagic suppurative pancreatitis. The three cases that I have to bring before the Society this evening are cases of diabetes which would seem to date their origin from a preceding attack of acute pancreatic disease. In all the cases this is an inference rather than a proved fact, inasmuch as in none of the cases has a post-mortem examination been made. Two of the cases resemble one another closely, but the third is, in my experience, unique. Full observations were only made in one case of a patient under my care at University College Hospital, the other two cases were seen in consultation, and for a variety of reasons records are imperfect.

Elizabeth D—, æt. 62 years, was admitted in April, 1903, to University College Hospital under the care of a colleague, complaining of vomiting, pain in the abdomen, and constipation. The

* At the Medical Society of London.

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patient had been subject to attacks of vomiting and indigestion for some months. For three weeks before admission the symptoms became more pronounced and she suffered from obstinate constipation. The patient had not previously suffered from any serious illness, and there was no history of phthisis in the family. On admission her respirations were 30, pulse 104, temperature 99° F., and on examination general distension and irritability of the abdomen were present, and a rounded swelling was to be seen and felt in the epigastrium and to the left of the middle line. On palpation the mass was very hard, but had no definite margins, and on light percussion yielded a resonant note. On deep percussion the resonance was impaired. Resonance was readily detected above and below the mass. The mass did not move with respiration. No fluid thrill was found in the abdomen, but there was slight impairment of resonance in the flanks which did not alter with change of posture. The liver, spleen, and kidneys could not be felt. The urine was acid with specific gravity of 1011, contained a trace of albumen but no sugar. No physical signs of disease were found in the chest and the heart was normal. During the next few days the patient was treated by enemata, and copious normal motions were thereby maintained and no vomiting occurred, but she suffered very considerable pain over the situation of the mass. This became so severe that an operation was advised on account of the pain. A vertical incision, three inches in length, above the umbilicus was made and the peritoneum was opened. A hard mass, firmly adherent posteriorly to the vertebral column and anteriorly to the stomach, was found in the region of the pancreas. It was quite immovable, and there were a number of adhesions between the mass, the stomach, transverse colon, and intestines. On inspection of the omental fat, numerous small, white, apparently caseous masses were observed. No further interference was deemed advisable and the wound was closed. The patient made a good recovery, and left the hospital free from pain and vomiting on May 6th. At the time of her discharge the mass was still present and had apparently undergone no change. At the time of the operation it was thought possible that the mass consisted of retro-peritoneal tuberculous glands with secondary infection of the mesentery. The patient was re-admitted under my care on October 15th, 1906, complaining of frequency of micturition, great thirst, and an irritating eruption over the whole of the perinæum. The frequency of micturition and the thirst had come on six months previously. The patient is greatly emaciated, the skin is rough and parchment-like, and there is very extensive pruritus about the anus and vulva. There were no physical signs in the chest. In the abdomen the scar of the operation performed three years ago reaches from one inch below the ensiform to the central point of the abdomen. A large umbilical hernia is present owing to separation of the recti, and hence palpation of the abdominal contents is rendered remarkably easy. The bifurcation of the aorta, the ribs, and the liver are readily felt through the thin skin and subcutaneous tissue. No tumour can now be detected, although it is easy, owing to the patient's emaciation and the condition of the abdominal wall, to feel the front of the vertebral column. The patient passes as much as 146 ounces of urine in the twenty-four hours, with a specific gravity of 1036, and containing 9 ounces of sugar per diem. Her weight on admission was only 5 st. 3 lb.

The case presented all the features of a moderately severe form of diabetes mellitus. She was treated by diet and discharged on December 8th, having gained one stone in weight and the percentage of sugar having diminished from some 6 per cent. on admission to ·9 per cent., and the specific gravity fell from 1036 to 1025.

The point of interest with reference to this case lies in the fact that on her second admission she presented all the signs and symptoms of a typical case of diabetes mellitus, but that three years previously she had suffered from a large tumour in the region of the pancreas which had disappeared spontaneously to an exploratory incision.

The second case seen in consultation was an almost identical one in that a lady had come under observation with a large abdominal tumour in the epigastric region which was explored by a laparotomy, regarded as malignant and incapable of removal. Subsequently to this symptoms of indigestion, thought to be dependent on pyloric obstruction produced by the mass, developed, and a second operation was performed and gastro-enterostomy effected. I saw the patient eighteen months after this second operation, when she was suffering from diabetes mellitus in a very severe form.

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Palpation of the abdomen, which was also easy owing to the emaciation, failed to reveal the presence of any tumour. In this second case the diabetes was so severe that diet had but little influence on the course of the malady.

I have during the last few years seen three other cases in which a mass was present in the epigastric region, and where an operation has been performed, and what was apparently a malignant growth found, and where, subsequently to the operation, the mass has disappeared and the patient regained health. But in none of these other cases, so far as I know, has glycosuria or diabetes ensued. The suggestion I venture to make is that in these cases we have to deal with a subacute pancreatitis accompanied with considerable swelling and induration of the organ. In the first case recorded, the foci seen at the operation in the omentum and looked upon as caseous were possibly areas of fat necrosis. In the lack of post-mortem evidence as to the state of the pancreas in these two cases it it is obvious that this view is only a theory.

The third case I wish to bring before you is that of a lady, æt. 31 years, who had an attack of what was looked upon as catarrhal jaundice presenting all the typical features of this affection. During its subsidence she was suffering with intense thirst and polyuria, and the urine contained as much as eight ounces of sugar in the twenty-four hours. In this case the symptoms of diabetes came on suddenly one night whilst the patient was under observation, and the urine was known to be free from sugar. Dietetic treatment reduced the quantity of sugar, but six months later the patient still required a rigid diet in order for the urine to remain free. The suggestion has been made that some cases of catarrhal jaundice are dependent on acute swelling of the head of the pancreas, and it is conceivable that this was the cause of this patient's illness. In some instances of catarrhal jaundice hepatic cirrhosis is shown to develop as a complication, and has been regarded as depending on an ascending infection from the biliary ducts. Diabetes has also been regarded by some as depending on an ascending infection of the pancreas from the onset, and I bring forward this case as possibly affording some support to this view.

Dr. F. W. Pavy: I am very happy to rise and speak before the Society, but I do not know that the cases which have been brought forward admit of very much discussion, taking the cases themselves. There can be no doubt that the disease, diabetes, is associated with the pancreas. The operations which have been performed upon animals, and likewise the observations which have been made upon the human subject, the number of cases which have been recorded in which diabetes has been associated with pancreatic disease, can leave no doubt in the mind that the pancreas is associated with changes occurring in connection with the assimilation or with the disposal of carbohydrates in the body. Seeing that in the experimentation, if the pancreas is removed to an extent leaving a small portion, that there is no diabetes, and that if this small portion which has been left is removed there is immediately diabetes, and invariably diabetes, there may be no question left, I think, that the pancreas is in some way or another associated with the assimilation or the disposal of carbohydrates in the body. I think we must look to enzyme action, which is a great deal to the front at the present time. There are enzymes connected with the building up of the complex molecules of which our bodies consist, and there are likewise enzymes associated with the breaking down of these molecules. In the first place it seems that the error is connected with the building up. If carbohydrate matter, taken as food, be properly built up into the large proteid molecule, it is safe from escape with the urine. I hold that if the small molecular body, sugar, is allowed to enter the circulation, you cannot possibly have anything else than its escape with the urine. It seems to me totally impossible that small molecular bodies, like sugar, getting into the circulation could be prevented from escaping with the urine. There is no such thing as impermeability of the kidney to sugar. Small molecular bodies, no matter from what source, when circulating through the kidney escape with the urine. Do we not notice, with regard to drugs and salines and so on, that if these enter the circulation they are to be found in the urine? Where water goes a molecular body not far different in size will follow. There can be no doubt that sugar reaching the circulation cannot be prevented from escaping with the urine. What is wanted is that the sugar should be taken into combination, should be built up by the assimilative process into a large proteid molecule, and this proteid molecule is safe

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from escaping with the urine under a natural condition of the kidney. When built up into this molecule the sugar is locked up, but it is ready to be taken by the tissues, just the same as oxygen. We find some free oxygen in the serum of the blood, but the greater portion of the oxygen goes into combination in the red corpuscles under the form of oxy-hæmoglobin, and in this form it is carried to the tissues, and the tissues take it from the combination, leaving the hæmoglobin free to take up further oxygen when it goes back to the lungs. So, it seems to me, is the case with regard to sugar, namely, that it is taken up into the proteid molecule by the assimilative process and thence carried to the tissues, where it may be taken off. The greater portion of it goes as a side chain, and is ready to be taken off by the tissues in proportion as it is wanted. It may seem that I am wandering from the subject of the paper, but I think you will find that it is not so, because I look upon the pancreas as the organ supplying a portion of the enzyme which is concerned in the assimilation or the building up of the sugar taken as food into the proteid molecule. We have enzymes concerned in both processes, the building up and the breaking down, and the evidence tends to show that the pancreas supplies a portion of the enzyme concerned. We now know that enzymes consist of two portions. There is a plasma portion or nitrogenous portion, but this is not effective alone; it requires the assistance of an activator. It is open to demonstration that neither of these constituents will act alone. For instance, taking zymose, the enzyme which breaks up sugar into alcohol and carbonic acid, as an illustration, it has been clearly shown that it consists of two portions: one of them non-diffusible, precipitable by alcohol, and susceptible of destruction by heat; and the other, the co-ferment, diffusible, not precipitable by alcohol, and not susceptible of being deprived of activity on exposure to a boiling temperature. The pancreas seems to supply the activator, and with the supply of this activator we may have an enzyme which is capable of causing the sugar molecule to be taken up by, or synthesised into, the proteid molecule. I think we have to look to the intestinal canal, that is to say to its mucous membrane, as constituting the seat of this action. The sugar is absorbed. In the first place, we have the process of food digestion, which is simply a preparatory process to absorption. Directly absorption occurs, assimilation must take place. If the assimilation here, or in the liver, does not take place, the sugar absorbed will pass into the general circulation and will be immediately revealed by the urine. So much is this the case, that within half an hour you may find sugar in the urine after the taking of carbohydrate which is not susceptible of being assimilated by the individual. For instance, taking the mild case of glycosuria, in this mild case a certain amount of carbohydrate can be assimilated. Keep within that amount of carbohydrate, and there is no sugar in the urine. But overstep that amount and immediately sugar is to be discovered in the urine. Even the smallest quantity of carbohydrate is sufficient to do this. I had a patient a few days ago whose urine was usually free from sugar. He is upon a restricted diet. He had taken on his own account, for breakfast, a small piece of toast. He did it to see whether it would be found permissible for him to advance to this change of food. He brought three specimens of urine; one of them was passed on going to bed, one just as he rose in the morning before breakfast, and the other about an hour after breakfast. The first two were free from sugar. The specimen which was passed an hour after taking the toast contained sugar. He came to me in the morning at 10.30, and at 10.45 I got him to pass more urine for examination. This was found to contain more sugar than the specimen passed an hour after breakfast. It seems to me that this shows that what ought rightly to occur is that the carbohydrate on absorption should be put into a state in which it will not be allowed to enter the circulation in the form of free sugar, that is to say, in the small molecular form belonging to sugar. And it seems to me also, from the evidence before us, that the pancreas is concerned in supplying the activator to the enzyme that is instrumental in leading to the carrying out of the assimilative or synthesising process. I have conducted experiments upon this matter, and I have found that on injecting sugar intra-venously into the system there is not only a rise in the sugar constituent of the blood in proportion to the quantity injected, but likewise a rise to a certain extent in the locked up carbohydrate of an amylose nature that is capable of being obtained on submission of the blood to the breaking-down influence of boiling with potash. As regards the sugar, it may be incidentally said that there is a certain standard small quantity normally present, it amounts to about 1 per 1000, which is independent of the character of the food, and

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which is revealed, notwithstanding the smallness of its amount, by passage into the urine. I have been trying for the last thirty or forty years to get it acknowledged by the profession that there is this output of sugar in normal urine, and it is only lately that it has begun to be generally realised as a fact. It is a point of fundamental importance, because under the nature of things if our carbohydrate food reached the general circulation as free sugar, it could not fail to proportionately pass out, as it does in diabetes, with the urine and thus become revealed. To revert to the amylose carbohydrate, I have said that it is to a certain extent increased in the blood by the introduction of sugar into the circulation, and I imagine that a portion of the sugar is absorbed by the bioplasm of the leucocytes, which constitute the living element of the blood, and fixed within them. Now, it happens that if with the sugar intra-venously injected some boiled extract of pancreas be conjoined, the figures for the amylose carbohydrate are carried to a higher level. Sugar possibly may be taken on by the bioplasmic molecule as a side chain, and become drawn off by the molecules of the tissues after the manner of oxygen in connection with hæmoglobin. In the natural process of assimilation following absorption from the alimentary canal, I conceive it is to the lymphocytes of the villi that we have to look as the agents concerned in carrying out the assimilation or synthesis of the carbohydrate of the food.

Dr. S. PHILLIPS: The two or three cases which Dr. Bradford recounted are somewhat similar to two cases which I recorded before this Society and the Clinical Society. They are two cases of pancreatic disease in which fatal diabetes occurred. One was that of a man who came into St. Mary's with symptoms usually described as being due to "catarrhal jaundice." He had slight swelling of the pancreas, and I thought he probably had acute pancreatitis. A surgeon examined him also and we felt sure of the swelling, and we were anxious to explore it, but the man refused. I kept him under observation and he was fairly well for nearly three years. He then came back to the hospital very ill with acute pain and considerable swelling of the pancreas. The day after coming in his temperature rose to 103° F. with a rigor, and sugar appeared in his urine. There had been no sugar in his urine up to that time. He soon died of diabetic coma. Post-mortem examination showed the head of the pancreas was very large; a recent hæmorrhage had occurred in it and a small abscess had formed. The hæmorrhage must have coincided with his acute attack of pain and suppuration, which was recent, and with his rigor. Though the disease was not as recent as in Dr. Bradford's case, it was a case in which a very recent attack of pancreatitis occurred on the top of a sclerosing pancreatitis which had commenced with an attack of jaundice. The other case was one which I recorded in the 'Clinical Society's Transactions' in 1903. A woman had jaundice; Mr. Owen explored the abdomen and found the head of the pancreas very large, probably malignant. She went out and then came back under my care six months afterwards. The jaundice had disappeared, but it was replaced by sugar in the urine. She very soon rapidly emaciated. No treatment, dietetic or otherwise, had any effect on the diabetes, and that is characteristic of pancreatic diabetes. She died of diabetic coma. Post-mortem examination showed she had a very much fibrosed and atrophied pancreas, and the head of it was not now enlarged, though it was known to be enlarged at the time of operation. As atrophy came on the jaundice was replaced by diabetes and coma. So I think those cases are somewhat similar to Dr. Bradford's, and the first, at any rate, tends to confirm the view that some of the cases which are recorded as catarrhal jaundice are really acute pancreatitis, and it has been suggested that some of these cases would do better if the gall-bladder were drained.

Dr. FOWLER said the cases which had been referred to recalled a paper written by Dr. White, of Philadelphia, entitled, "The Successful Result of Unsuccessful Operations." There are many such, and particularly in connection with the abdomen. Every surgeon now has had some experience of the opening of the abdomen and, finding a condition which apparently could not be relieved or interfered with, has closed the abdomen followed by the disappearance of the symptoms. I saw a case to-day in the hospital which may or may not be a similar case to Dr. Bradford's. It was that of a woman deeply pigmented. She recently had an attack of jaundice which had passed off. She has pigmentation round the eyes, a tumour in the neighbourhood of the pancreas, and glycosuria. She seems very well now, better than one would expect, and it will be interesting to see whether that tumour disappears or gets smaller. We are greatly indebted to Dr. Bradford for bringing these cases forward and to Dr. Pavy for his remarks.

Dr. BRADFORD, in reply, said: With reference to what Dr. Phillips recorded, the point of my cases is not so much that the diabetes was associated with probable pancreatic lesions—I should not have brought them forward for that—but rather the fact that there was evidence of the existence of probable pancreatic disease at a period antecedent to that at which the diabetes was present, and at a period when glycosuria was known to be absent. And in reference to the case in which jaundice was present, the diabetes there ensued rapidly, in the fourth week after the onset of the disease, and the catarrhal jaundice lasted three weeks, and during the first week of convalescence diabetes appeared suddenly, and in that case there was no pancreatic tumour.

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A LECTURE

ON

ASSOCIATED ABDOMINAL AND THORACIC DISEASE.*

By G. NEWTON PITT, M.A., M.D., F.R.C.P.,
Physician and Lecturer on Pathology, Guy's Hospital.

I HOPE to interest you this afternoon by bringing before you some of the associations which are not infrequently met with between lesions in the thoracic and abdominal cavities. Many of the symptoms which arise in connection with these lesions have to be very carefully considered, otherwise not infrequently we may go astray with regard to their origin. While we theoretically fully recognise the influence of one part of the body on the other, not infrequently in practice we fail to bear it in mind. There is a tendency, when the most obtrusive symptoms are obviously associated with one part of the body, to concentrate our attention on that organ and overlook the occurrence of active organic changes in other organs. Associated lesions may arise in different ways, and it is only by observation that we can detect them. An infective process may produce lesions in distant parts of the body which will differ from those at the primary seat. We must possess experience to enable us to track down the various symptoms to their correct pathology. For instance, a collection of pus in the pelvis, in the upper part of the peritoneal cavity, or in the pleura, may be productive of secondary infections which will differ in each of these cases according to their various anatomical surroundings. Although the peritoneal sac at a post-mortem examination is empty and we are apt to look upon it as of little importance, yet in life there is a constant circulation of fluid passing

* Delivered at the Medical Graduates' College and Polyclinic.

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through it, and active changes go on. Not only are the surfaces of the viscera kept smooth, but active phagocytic changes take place which keep the cavity sterile. Water and salts are freely absorbed by the veins and capillaries in the omentum, and in the walls of the intestine and of the abdomen. The channels of exit by which colloid materials, such as organisms, serum, and blood, can escape are limited to the under surface of the diaphragm. No absorption of these materials takes place through the abdominal wall or through the omentum, they only pass out through the stomata on the under surface of the diaphragm. When the diaphragm contracts it increases the intra-abdominal pressure, and these materials are forced upwards through the diaphragm. The effect of gravity is here of great importance, because it is mainly those particles which have already come close to the diaphragm which are affected by the muscular contraction. The circulation is kept up by the diaphragm acting as a suction pump, by first drawing the particles up from the lower towards the upper part of the peritoneal cavity, and when they come in contact with the diaphragm, squeezing them through. It has little effect, however, on the material in the lower part of the abdomen. This was strikingly proved by some experiments on dogs, in which particulate material had been injected into the lower part of the peritoneal cavity. When the animal was held with its head upwards, very little of the material passed out of the peritoneal cavity during the next forty-eight hours, whereas when the animal was placed head downwards, within five minutes some of the particulate material was found to have passed out of the abdominal cavity into the glands situated behind the sternum. It is only recently that the great influence of gravity in connection with circulation in the peritoneum has been insisted upon, and it is of extreme practical importance. It is essential also to bear in mind that at a post-mortem examination, when you are anxious to determine which are the organisms that have been responsible for peritonitis, the best way to obtain a pure culture is to take one from the glands behind the sternum rather than from the peritoneal cavity itself. So long as infective material remains in the peritoneal cavity it has no influence on the general system; it is only the material which has already been absorbed from the cavity and which has passed out that produces the toxic symptoms of peritonitis. The local changes which take place in the peritoneum indirectly affect the blood by causing fluid and cells to pass into it from the vessels, and they thus alter the arterial pressure. The toxic effects are limited to the material which has already left the peritoneum. There is, therefore, no constant relation between the symptoms which are present during life and the appearances in the peritoneum which are seen after death in cases of fatal peritonitis. What is seen in the peritoneum are the local signs of the lesion, the attempt which the peritoneum has made to keep the toxic material away from the rest of the body, whereas it is the toxic material which has been allowed to escape which has produced the fatal symptoms. Hence the very great difference there is in the fatality arising from perforations occurring in the upper part of the abdomen, such as gastric or duodenal ulcers, compared with that due to lesions in the lower part as in the pelvis or in the appendix. In the former case the material is early squeezed into the general circulation by the diaphragm, whereas in the latter it may remain stationary for a considerable time, and being localised it quickly loses its virulence. To save the life of a patient who has a perforated gastric ulcer it is necessary to operate in twenty-four hours, whereas in a case of appendicitis or pyosalpinx you may often wait for two or three days. It is very rare for inflammation of the pleura to set up peritonitis, whereas it is not infrequent for peritoneal inflammation to spread through the diaphragm and involve the pleura.

Associated thoracic and abdominal lesions may be grouped as follows:

(1) Thoracic lesions: pleural, pericardial and pulmonary, depending on (*a*) infection, (*b*) growth in abdomen.

(2) Multiple chronic serous effusions.

(3) Abdominal lesions and symptoms depending on (*a*) pulmonary, (*b*) cardiac disease.

(4) Some difficulties of diagnosis between abdominal and pulmonary disease.

(5) Associated digestive disturbances and pulmonary lesions.

(1) (*a*) Examination of a series of 62 cases of streptococcal empyemata showed that 14 of them, or between one-fourth and one-fifth, spread from the abdominal cavity, the origin of the others

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being pulmonary 25, chest wall 3, mediastinal 2, undetermined or so-called primary 13. It is owing to the mechanical conditions to which I have already referred that pleurisy only occasionally occurs in connection with pelvic peritonitis or appendicitis, and even then the infection does not take place very early; the virulence of the micro-organisms has been by that time so materially reduced that most of the cases do well, and it is only comparatively rarely that it is necessary to drain the chest. An examination of 700 consecutive cases of appendicitis shows that in about 1 per cent. of them pneumonia occurs, and in about 3 per cent. pleurisy. But if we limit ourselves to cases of suppurative appendicitis, the number of cases in which a pleuritic rub develops is probably nearly 10 per cent. There are two particular conditions in connection with appendicitis which may lead to pleurisy. The first is when there has been a general peritonitis, so that the whole cavity has been infected, and although the abdomen be opened and the appendix treated, it is not uncommon for the patient's condition, after improving for a time, to begin again to deteriorate, and not infrequently this is found to be due to a sub-diaphragmatic collection of pus with a secondary involvement of the pleura. So it is extremely important in order to as far as possible prevent fluid from reaching the upper part of the abdominal cavity, to mop out the upper part of the abdomen carefully, particularly in the region of the spleen and the liver when the upper part has been infected. After an operation for appendicitis the patient should be propped up in the sitting posture, as you are then more likely to be able to localise the infective material to the pelvis and the lower part of the abdomen and thus prevent general infection. It has been shown that cases of appendicitis may be propped up within less than twenty-four hours after the operation, with the greatest advantage. The second condition of appendicitis which is apt to lead to infection of the pleura is when the appendix lies behind the cæcum, and the inflammation has involved the retro-cæcal connective tissue. In such cases an abscess may form, the symptoms of which may be very obscure, partly because the abscess has been covered by the cæcum so that there is no dulness on percussion, and partly because it can spread up without much tension while there is but slight leucocytosis and very little pyrexia. These are the cases in which operation is often unduly delayed because of the late stage at which the symptoms manifest themselves, and it is in this group of cases particularly that the pleura as well as the under surface of the diaphragm is apt to become involved. The practical lesson, therefore, to be drawn is, that when you have a case of appendicitis which is not doing well, you should make it a rule to examine the condition of the pleura carefully, more particularly the right pleura, as not infrequently that will be found to be the seat of secondary infection. Other spots which require special attention where there has been general infection of the peritoneum are the pelvis and the under surface of the diaphragm, especially around the spleen and liver, where there is likely to be a collection of fluid unless you keep carefully on the look-out for it and prevent it. I shall not quote many cases, but I may mention a characteristic one illustrating these points.

A boy, æt. 18 years, was admitted on January 13th, under my care, having had a headache for four days, and vomiting for twenty-four hours. He was restless, and had abdominal pain with tenderness in the right iliac region, where a small, ill-defined mass was palpable. The abdomen was opened and an abscess was found burrowing up behind the cæcum and the retro-peritoneal tissue. At the time of the operation there was nothing to cause any grave anxiety. He progressed favourably for five days. But on the eighteenth day his temperature began to rise, and a rub was audible in his right axilla. During the next twenty-four hours he was very much worse, his wound was opened up and a freer drainage was established in the lumbar region. His right chest was also drained; several ounces of very foul sero-pus containing *Bacillus coli* were let out. In spite of this, although he was examined very carefully his general toxæmic condition increased, and he died on the 22nd. Post mortem we found there had been a small accumulation of foul pus in the pelvis, which we had not detected. But the chief reason he got worse was that there was also a collection of pus in the form of a sub-diaphragmatic abscess which had formed above and behind the liver. This is typical of how a sub-diaphragmatic abscess forms, and of the way in which the pleura becomes infected.

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Another initial abdominal lesion which may cause pleurisy is pelvic peritonitis, which is of importance because it is so common, and although pleurisy does not very frequently result, still this cause is by no means negligible. Infection originating in gall-stones, especially when there is local suppuration in connection with the bladder, readily infects the pleura. An hepatic abscess not infrequently opens into the lung, and as soon as the pus reaches the upper surface of the liver it is fairly certain to cause pleurisy. Laparotomies, especially gastric operations, may be the origin of pleural infection.

Cases of sub-diaphragmatic abscess are of considerable interest. They are somewhat infrequent and the symptoms are obscure. These cases are easily mistaken for pleuritic effusion, which is often present as well, and in cases of effusion into the chest an exceptional dulness in front should raise the suspicion that the fluid may not be entirely above the diaphragm. Such an abscess may originate with a perforated gastric or duodenal ulcer, where the perforation has been small and the peritoneal cavity has not been flooded with gastric contents so that the collection has had time to become encysted, especially when the perforation has been on the posterior surface opening on to connective tissue. Sub-diaphragmatic collections of pus may also start from an hepatic abscess or a suppurating hydatid, from inflammation associated with gall-stones, from appendicitis, and not infrequently from a widespread diffuse peritonitis, from whatever cause, when by means of surgical treatment the patient's life has been saved, and yet a small residual collection of pus has formed there which it has not been possible to drain. The most common places for sub-diaphragmatic abscesses to become encysted are between the liver and the diaphragm, or on the left between the spleen, stomach and diaphragm. In either case they quickly lead to infection of the pleura above and more rarely to infection of the pericardium. One of the most characteristic signs is when two fluids presenting different characters have been drawn off when aspirating the chest. In such cases one will be found to have been drawn off from above and the other from below the diaphragm. As a rule the fluid above the diaphragm is much less turbid and is thinner in consistency than that withdrawn from below. The following was a very unusual case of a sub-diaphragmatic collection of fluid. A boy, æt. 9 years, was run over by a cart, and after a month's residence in the surgical wards he was transferred to my care, with ascites and slight jaundice. The ascites increased in amount, and on two occasions ten pints of bile-stained fluid were drawn off. We came to the conclusion that the boy had an encysted sub-diaphragmatic collection of fluid, which we drained. Later on pyo-pneumothorax developed and the boy ultimately died. Post mortem we found a large depressed scar on the surface of the liver the result of an extensive laceration, bile and serum had been poured out and formed a localised collection. The changes, however, had gone on so slowly that the surgeon determined not to explore the abdomen. It was not until later on when we had evidence of a definite sub-diaphragmatic collection that we decided to explore. But the combination of pyo-pneumothorax and the sub-diaphragmatic collection proved fatal. Many other examples of pleurisy and empyema secondary to local abdominal inflammations might be given. To illustrate another cause we may cite the case of a man, æt. 33 years, who came in with an empyema, which was successfully drained, and it was only some three months later that it was obvious that it had originated from a peri-nephritic abscess which then required surgical treatment. The empyema had at first dominated the situation and the peri-nephric trouble was overlooked. We recently had a case of pleurisy secondary to cholecystitis. The man, æt. 40 years, had had frequent attacks of hepatic colic, and was admitted with pyrexia and very severe pain in the region of the gall-bladder; shortly after a pleuritic rub was audible over the right lung in front. Ultimately it quieted down and he did well without operation. Pneumonia is found much less frequently than pleurisy, in connection with abdominal inflammation; still such cases do occur. For instance, a boy, æt. 13 years, was admitted under my care on the 25th of last month with appendicitis, which he had had for twenty-four hours. The pulse during the night following his admission ranged from 84 to 124, and the temperature was between 100° and 101° F.; there was no vomiting, and his chest was quite normal. The appendix, which was found to be embedded in a mass of omentum, was removed,

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and a small abscess containing a couple of drachms of pus was opened. The boy, however, three days later had not improved; his temperature ran up; we found dulness at the right base, with bronchial breathing due to the development of pneumonia. The temperature ran the ordinary course in that disease, and he ultimately made a very good recovery. Apparently the pneumonia did not very materially increase the risk to his life.

Occasionally the abdominal mischief may also spread to the pericardium, as in the following case of sub-diaphragmatic abscess, which set up pericarditis as well as an empyema. The patient was a woman, æt. 34 years, who had been laid up at home complaining of intense abdominal pain. A fortnight later she was admitted with acute pericarditis and there was effusion of fluid into the left chest. After careful examination we came to the conclusion that she had also a perforated gastric ulcer. The history of the symptoms was characteristic. When the gastric ulcer is situated anteriorly the patient very often dies within twenty-four or forty-eight hours. It is when the abscess burrows into the connective tissue and becomes encysted, so that the fluid is enabled to collect, that the patient is not killed at once. In this instance the patient lived a fortnight before she came under observation. She then had a well-marked sub-diaphragmatic abscess presenting in the epigastrium, a to-and-fro pericardial rub, and there was a well marked *bruit d'airain* over the sac which presented in front, which was undoubtedly transmitted from the stomach, although the sac itself gave a dull note and was full of fluid. The left chest was explored and thirty ounces of thin, foul fluid, containing *Bacillus coli*, were let out. The subdiaphragmatic abscess was opened and another twenty-five ounces of thick foul pus was let out. The patient improved for a time, but died at the end of a fortnight. We found post-mortem that there was another small collection of pus behind the spleen which could not be got at during life. That is one great difficulty in these cases; there is often a collection of material in the posterior part of the abdomen which one fails to drain efficiently.

I need only refer briefly to pulmonary embolism. This may not infrequently result from venous thrombosis in the abdomen.

(1) (*b*) The next group is that of *malignant disease*, as a result of which a patient may present signs both of ascites and pleuritic effusion. They are not very common, yet I have notes of some seven cases where pleuritic effusion was found to have originated from cancer of the stomach. We may refer to the case of a man, æt. 34 years, who had double pleuritic effusion together with ascites. When admitted there was dulness up to the sixth and seventh ribs on both sides, and great distension of the abdomen. The chest and abdomen were tapped on several occasions with great temporary relief, but he finally died. We then found what we had not suspected, viz. a primary malignant growth in the stomach with secondary nodules scattered over the pleuræ, pericardium, and peritoneum. We had recognised that he had malignant disease, but we had not realised that it had started in the stomach. To quote another: a man æt. 45 years came under my care with a right pleuritic effusion, which recurred three times after tapping. At first the fluid was serous, but it was not until we noticed the enlargement of the sterno-mastoid gland on the left side that the seriousness of the mischief was realised and we appreciated that it was a case of malignant disease which ultimately turned out to have originated in his pancreas.

The glands behind the sterno-mastoid are of great assistance in the diagnosis of abdominal cancer. Twenty years ago Troissier drew attention to the fact that very often the first positive indication of malignant disease in the abdomen may be the presence of a growth in the gland which is situated behind the clavicular end of the left sterno-mastoid. This gland is found to be involved four times as frequently as the right one, and indicates malignant disease occurring generally in the upper part of the abdomen, particularly in the stomach; but it may be in the pancreas or even in the peritoneum. It may also occur in connection with the œsophagus and mediastinum, so it is not conclusive of any one organ being involved. Still, it most commonly indicates the stomach. Malignant disease in the abdomen may spread upwards directly by contiguity until it reaches the under surface of the diaphragm. Growing through the diaphragm it may involve the upper surface of the pleura, and in that way spread up along the glands of the mediastinum but independently of that, it may spread by the lymphatics into the chest and up to the neck. The lymphatics of the stomach may be

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divided up into three groups. There are the coronary vessels, which run to the lymphatic glands behind the pancreas; there are the gastro-epiploic which run up to the thoracic duct, and from the thoracic duct extend up and involve this gland in the neck; and the third group, the right gastro-epiploic, run behind the duodenum to the right side of the diaphragm, and in that way infect the right side of the pleura. Malignant disease of the stomach, then, can, by growing up along the lymphatics, spread up the thoracic duct to this gland on the left side, or it may spread up, but much less frequently, to the right side. I published some cases two years ago showing the importance of this gland in malignant disease, and only a fortnight ago Dr. Mitchell Stevens gave a very excellent paper which was published in the 'British Medical Journal,' February 9th, 1907, dealing with the same subject, and he pointed out that in many cases the growth extends continuously along the duct, in this way corroborating the view which Mr. Handley put forward in his Hunterian Lecture at the College of Surgeons last year, showing that malignant disease spreads along the lymphatics by solid bundles of growth, and not merely by emboli. It used to be supposed that cells were carried singly or in small clumps as emboli, but Handley showed microscopically that you can trace solid bundles of growth along the lymphatics, until the larger vessels are reached, and this is in accordance with the view Dr. Stevens put forward in the article to which I have referred. Another patient illustrating this died last week. He was 45 years of age, and came in with a mass of glands involving the posterior triangle on the right side of the neck, and extending down along the trachea. Examination showed evidence of a mass of growth in the anterior mediastinum, invading the root of the right lung. Later on we were able to detect a mass of growth in the epigastrium, which became gradually more obvious, and post mortem the growth was found to have originated in the head of the pancreas.

(2) The next group consists of cases of *multiple chronic serous effusions*, which occur under several different conditions. Such effusions occur in the later stages of chronic cardiac disease, with malignant disease and with obvious tubercle. To these I do not now propose particularly to refer, but rather to those cases which were often termed simple peritonitis and simple pleurisy, which really meant those forms about which we knew least, but which we now begin to recognise are tuberculous.

I shall not distinguish between the so-called simple and the tuberculous cases. Simple cases of peritonitis, as a rule, when explored do not present any obvious tubercles on the serous membranes, but in many cases it is possible by inoculating animals to produce tuberculosis. Undoubtedly many of the cases do own tubercle as their origin. The condition has been described more particularly in Italy, as polyorrhomenitis. The chronic inflammation may involve one or more of the serous sacs, the peritoneum, pleura, pericardium, but most commonly the peritoneum and both pleuræ. The occurrence of ascites due to chronic peritonitis is about half as frequent as that from cirrhosis of the liver. It is noteworthy that these patients often live for a long time and may be tapped on many occasions, whereas it is comparatively rare to meet with prolonged ascites from cirrhosis of the liver; it would appear probable that fluid only accumulates in chronic peritonitis when the stomata on the under surface of the diaphragm have been blocked up by cells. The secreting surface is probably constantly pouring in its fluid, and the stomata in the diaphragm gradually become blocked up. When such cases are tapped you will find that the rate of accumulation of the fluid gradually decreases. In a case under my care at the present time, last autumn it was retained at the rate of 10 oz. a day. A little while later the increase was at the rate of 9 oz. a day, and now the rate of increase is about 8 oz. a day, the reason being that the serous surfaces gradually become adherent and the amount of secreting surface is diminished. (Later on the re-accumulation of fluid gradually ceased and the patient went out in very fair health.) Besides these there are also cases of chronic indurative mediastino-pericarditis, which are generally tuberculous in origin when great thickening of the mediastinum also takes place. The pericardium becomes adherent and very often the pleuræ; the illness is characterised by general anasarca, the most extreme cyanosis with fluid effusions in the various cavities; the condition ultimately is fatal, but often it is not fully and accurately diagnosed during life.

(4) As an illustration of the difficulties of diagnosis

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I have seen more than one case of pneumonia in which, for some days, we have been in doubt as to whether we were dealing with acute peritonitis, or with a commencing consolidation of the lung. On the other hand, some years ago I had a case of croupous pneumonia in which suppurative pneumococcal peritonitis developed, which we failed to recognise as it gave rise to no symptoms that attracted our attention. This is almost the only condition in which inflammation of the peritoneum results from inflammation of the chest. In cases where there is a doubt between pneumonia and abdominal disease, the history, the disturbed pulse, and respiration ratio, the temperature, and generally, the presence of a patch of fine crepitations with deficient breathing, which will later be replaced by bronchial breathing, will generally enable a correct diagnosis to be made. A considerable number of the cases of pneumonia, probably one fourth, commence with symptoms of digestive disturbance, and not infrequently with diarrhœa and vomiting. In these cases, particularly when the physical signs are delayed two or three days, there may be very great doubt as to whether the main lesion is not abdominal: *e.g.* a boy was admitted into the hospital with severe pain in the region of the appendix. The temperature was 103° F., respirations 44, and the pulse very rapid—120. The right leg was drawn up, the right iliac region was tender, that is to say there was distinct hyperæsthesia. The abdomen, however, was supple; there were no physical signs in the chest; leucocyte count 23,000 per c.mm. The abdomen was opened under the impression that it was probably a case of appendicitis; the appendix was found to be perfectly normal. Next day the physical signs in chest still remained normal, and it was not until forty-eight hours afterwards that there was slight impairment of the note at the right base, and the following day pneumonia developed. That, therefore, was a case which presented the symptoms of appendicitis, although it was really a case of pneumonia. As an illustration of the converse a patient was admitted with a history of having had acute abdominal pain for twelve hours, with rapid respirations; a rub was audible over the lower part of the left lung, and the abdomen was moderately rigid. The conclusion in that case, chiefly on account of the rub, was that the illness was a commencing pneumonia. She was allowed by the house physician to wait until the morning, when it was discovered she had a perforated gastric ulcer, by which time, however, she had become so much worse that it was impossible to operate. In these cases, therefore, you have to make up your mind at once, very often on imperfect evidence, and occasionally you must make mistakes. But it is generally wiser to err on the side of opening the abdomen in a case of pneumonia, as may very exceptionally happen, rather than to allow a gastric ulcer which has perforated to be left without operation.

Cases of abdominal trouble which at first present symptoms of thoracic disease, I would refer to pneumonia and typhoid. Fortunately in this country it is very rare for typhoid cases to present evidences of pneumonia, but in the South African War it was a very common condition, and I have notes of two recent hospital patients who came in with pneumonia who ultimately turned out to have also typhoid, the consolidation of the lung being due to the typhoid.

(5) Next I would refer to the fatal effects of over-feeding in cases of acute pneumonia and in severe heart disease. It is not infrequent at a post-mortem examination to find in a fatal case of pneumonia or heart disease that the stomach is enormously distended with large quantities of egg and milk, often in the form of large clots, and it also contains much gas, due to the misdirected energies of the nurse. There is no doubt that in the severe stages of these diseases far better results will be obtained by keeping the patient on water and stopping food than by overloading the stomach with food which it is incapable of digesting

In conclusion, as to errors in diet producing thoracic disease. There is no question in my mind that almost all the bronchitis, and much of the broncho-pneumonia in babies, is entirely due to bad feeding, and Dr. Rabagliati has pointed out that in the Midlands, where the working people live largely on bread and butter and tea, a great deal of the chronic bronchitis from which they suffer is due to their defective diet. And its occurrence may also, in a large measure, be prevented by suitably altering their diet. The influence of diet in the production of asthma is also a matter of importance.

I hope I have been able to put before you a sufficient number of instances to show the close communication and association there is between lesions above and below the diaphragm, and the chief lesson I would like to inculcate would be that we are very apt to concentrate our attention on some prominent symptom, and fail to notice changes which are going on in distant parts of the body, and it is only by systematically examining the whole of the body that we can save ourselves from making errors of diagnosis which with care ought to have been avoided.

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THE SURGERY OF THE CUTANEOUS MELANOMATA.

By W. SAMPSON HANDLEY, M.S.Lond., F.R.C.S.,
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THE melanotic tumours of the skin are divisible into two classes, simple and malignant melanomata. The feature which marks off a melanoma from all other tumours is the deposition within its cells of fine granules of a brown or black pigment—melanin. Although this pigment when fully developed is iron-free, it may in its young state give the microchemical reactions for iron, a fact which points to its elaboration from the pigment of the blood. It is of practical importance to note that melanin, though a stable and inert substance, is bleached by chlorine, for this reaction enables it to be distinguished from the carbon particles which are so often found, not only in the glands of the thorax, but in those of the abdomen and the neck.

It is a significant fact that melanomata only arise in situations where melanin may be found in the normal body, that is to say, in the skin, upon mucous membranes near their junction with the skin, more especially at the mouth, the anus or the vulva, in the choroid coat of the eye, and in the spinal pia-mater. Rarely melanomata may arise in connection with the skin of ovarian dermoids.

The chromatophores.—In the normal body the production of melanin is the exclusive privilege of a certain class of mesoblastic cells, known as chromatophores. These cells are specialised connective-tissue cells, fusiform or stellate in form, and characterised especially by the possession of two or more branching processes, several times the diameter of the cell in length. In the skin the chromatophores are found normally between the fibrous bundles of the superficial layers of the dermis. The pigmentation of the deeper layers of the cutaneous epithelium in the negro appears to be due to the intrusion between the epithelial cells of long branching processes derived from the chromatophores of the subjacent dermis.

The pigmented cells of simple melanomata may often be clearly recognised as typical stellate chromatophores, and the cells of a malignant melanoma are also of mesoblastic origin, and represent chromatophores in various stages of embryonic development or senile decay. But in the malignant melanomata the cells may lack the characteristic stellate form and branching processes, and large areas of the tumour may be entirely devoid of pigment. Under these conditions the tumour-cells may closely resemble cells of epithelial origin, and consequently certain of the malignant melanomata have been described by some authors as carcinomata. Although this opinion is widely held, it is, I believe, erroneous.

The simple melanomata are of congenital origin, or first appear during early infancy. They are probably the commonest of the tumours that affect the human body. Clinically their variations in size and appearance are extreme. They are frequently multiple. By the Germans they are all included under the term "pigmented nævi," the word "nævus" simply indicating a birth-mark or congenital malformation of a localised area of skin. The name is a good one, for there is a close relation between the nævi which consist chiefly of vessels and the melanomata. A malignant melanoma may sometimes originate from a vascular nævus which appears devoid of pigment.

The following clinical varieties of simple melanoma have been described:

(1) Blue nævi—tiny spots like gunpowder tattooing, the colour of which does not disappear on pressure. Their blue colour depends on the situation of the pigment somewhat more deeply than usual in the dermis (Tièche).

(2) Hairy moles—slightly raised brown or black patches, covered with hair. They may be of any size, the largest covering a considerable part of the surface of the body.

(3) Glabrous moles—brown or black patches, or non-pigmented soft fibrous plaques, devoid of hair.

(4) Pigmented warts and warty moles. These are similar to Classes 2 and 3, but are more definitely raised above the general surface by hypertrophy of the cutaneous papillæ.

The structure of simple melanomata.—In spite of their widely differing appearances, all the simple melanomata are built upon a common plan. Their characteristic feature is a richly cellular layer intercalated in the fibrous structure of the true skin or dermis near to its surface of contact with

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the epidermal epithelium, but often separated from the epithelium by a thin lamella of the normal fibrous structure of the dermis. This characteristic layer closely resembles in structure an alveolar sarcoma. It consists of fibrous bundles between which lie groups and lines of richly protoplasmic, polygonal, non-pigmented cells, which are to be looked upon as embryonic chromatophores. To these cells the name "nævus cells" has been applied. The pigment cells or chromatophores of a mole are generally found superficial to the nævus cell layer just beneath the epithelium; they are stellate or fusiform, or have degenerated into large shapeless masses of melanin. Sometimes the pigment is also found in the deepest layers of the epithelium, or appears to be confined entirely to this layer. This appearance is probably a fallacious one, due to the intrusion between the epithelial cells of branching processes of the chromatophores of the underlying dermis, richly laden with melanin granules.

The hypertrophy of the cutaneous papillæ which distinguishes a pigmented wart appears to be merely a secondary change, probably dependent on an obstruction to the lymphatic circulation.

The treatment of simple melanomata.—There are two rules in the treatment of simple melanomata which should only be broken with great hesitation. Caustics should never be applied to them, and the tumour should never be cut into. Care must be taken that the tumour is not subjected to chronic irritation, as by picking and scratching, or by the pressure of the clothes. By far the larger number of simple melanomata require no treatment whatever and should be let alone. In the following circumstances excision becomes advisable:

(1) If the tumour is prominent, or of considerable size, even though it is not growing.

(2) If its situation renders chronic irritation likely or unavoidable. Thus in a man a melanoma of the inner side of the thigh is constantly subject to the friction of the clothes. A melanoma of the wrist, or of the neck of the vulva, or of the anal region for one reason or another come into the same category and should be removed, together with the whole thickness of the skin from which they spring, and a narrow margin of the surrounding healthy skin.

Under the following conditions excision is imperative:

(1) When the mole displays a tendency to ulcerate with the formation of crusts or scabs on its surface.

(2) When bleeding takes place from its surface.

(3) When the mole is increasing in area or becoming more prominent.

If the increase in size is rapid or considerable, and especially if there is palpable enlargement of the lymphatic glands, the mole must be treated as a malignant melanoma, a condition which demands a serious and carefully planned operation, not the mere snipping-out of the tumour.

If the mole is so large that the edges of the gap left by excision cannot be brought together, immediate skin grafting should be practised.

The most difficult cases of simple melanoma to treat are those in which the large area of the mole makes excision impossible. For example, a hairy mole may almost cover the face, producing an appalling disfigurement. For such cases depilation by X rays has been recommended as a palliative, but of course the pigmentation of the skin remains. In these difficult cases it is legitimate, if the patient wishes it, to depart from the usual rule not to cut through the substance of a mole. The pigmented skin may be spoke-shaved with a sharp razor until the deeper unpigmented layers of the dermis are exposed. The raw surface is then covered with a large graft (single, if possible) of healthy skin. It may be advisable to carry out the method by a series of operations, not all at once. There is undoubtedly some risk of exciting malignant change in the mole, and these very large moles should only be touched when their unsightliness threatens the career and prospects of the patient.

Malignant melanoma of the skin.—Malignant melanomata of the skin may be divided into two classes:

(*a*) Those arising from pre-existing simple melanomata.

(*b*) Implantation melanomata.

The second class is by far the less numerous, but the more interesting. It includes such cases as those recorded by Mr. F. S. Eve, where, after an interval of some years, a malignant melanoma developed in the site of a punctured wound of the palm, or in the site of a punctured wound of the sole of the foot. This variety of melanoma has a predilection for positions liable to injury, and par-

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ticularly for the heel and for the paronychial skin of the fingers and toes, especially upon the index finger, and upon the outer side of the great toe. Mr. Jonathan Hutchinson has described these paronychial growths under the name "melanotic whitlow." They have occurred with especial frequency at the London Hospital, which serves the tailoring district of the East End, in women, and upon the index finger, facts that go to show that needle pricks and other injuries of the paronychial skin are an essential factor in their etiology. In some cases the growth is preceded by a septic paronychial whitlow of the ordinary kind. The inflammation subsides, but a firm swelling remains at the side of the nail, not pigmented, and resembling a chronic inflammatory swelling. Mr. Hutchinson describes a narrow black border round the base of the swelling which enables its true nature to be recognised. The glands and other metastases of these growths may show an intense black pigmentation in strange contrast to the faintly-pigmented primary growth. In regard to paronychial melanomata of the great toe their occurrence to the outer side of the nail shows that the sharp edge of an ingrowing toe-nail is the weapon concerned. Implantation melanomata occur also in the lowest portion of the rectum, and here punctured injuries by fish-bones are the probable cause.

How do punctured wounds produce malignant melanoma? Mr. Bland-Sutton has drawn attention to the implantation cysts, lined by epithelium, which originate from particles of epithelium carried down into the deeper tissues by a sharp instrument. I believe that the melanomata which originate in the scar of a puncture, and in general those which arise apart from a pre-existing simple melanoma, are due to the traumatic implantation into the subcutaneous tissues of a group of the chromatophores which are normally found in the overlying dermis. It is for this reason that I have given to these tumours the name "implantation melanoma."

The *diagnosis* between a simple and a malignant melanoma may be very difficult, and in case of doubt the tumour should be treated as malignant. Rapid increase in size, the onset of ulceration and hæmorrhage, above all the presence of enlarged glands, these symptoms, or any one of them, should be taken to indicate malignant degeneration of a mole. But the distinction between a dangerous simple mole and one which is already malignant is very difficult to draw sharply.

Clinical course.—Melanotic sarcoma is not usually a painful disease, nor, except in its later stages, does it as a rule produce marked constitutional symptoms. It is not so rapidly fatal as is generally thought, and the average expectation of life after the onset of malignancy verges on three years. Frequently the patient is able, with a struggle, to do his ordinary work until within a few days or weeks of the end, although emaciation and abdominal enlargement, due to ascites and to the hepatic deposits, show that the finger of death is upon him. The primary growth or the secondary subcutaneous nodules may, however, when ulcerated, be the seat of severe pain in a relatively early stage.

During the course of a malignant melanoma masses of tumour tissue may undergo disintegration and may even completely disappear. Under such circumstances melanin granules in considerable quantity may be set free in the fluids of the body, and may thus produce a diffuse pigmentation of the skin and of the deeper tissues. The excretion of these pigment granules by the kidney may produce a dark coloration of the urine—melanuria. I have seen the glomerular tufts of the kidney obstructed by embolised masses of pigment, apart from any evidence of true secondary deposits in those organs. In places the pigment masses had broken through into Bowman's capsule.

The mode of spread of malignant melanoma.—From an operative point of view it does not much matter how a malignant melanoma originates. But it is absolutely essential that the surgeon who sets out to excise a malignant growth should know precisely how the growth spreads. An investigation of this question has convinced me that the process of lymphatic permeation, which I showed to be a main factor in the dissemination of breast cancer, takes a very important share in the spread of malignant melanoma.

The lymphatics of the skin.—Lying in the subcutaneous fat, upon or close to the deep fascia, is a plexus of fine lymph-vessels, which forms a complete investiture for the whole surface of the body. This great fascial plexus is drained by various sets of large trunk lymphatics into the cervical, the axillary, or the inguinal glands. It receives its lymph from a number of tributaries, each of which takes its origin

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in a convergent bundle of lymphatic capillaries. Most of these tributaries originate from the skin and dip down vertically from the deep aspect of the dermis to join the plexus. Others, originating in the muscles and other deeper tissues, join the fascial plexus on its deep aspect.

The beginnings of dissemination.—When a simple melanoma becomes malignant the mass of actively proliferating cells exerts a considerable pressure on the surrounding tissues, comparable to the force of growth which sometimes enables a fungus to lift the paving stone beneath which it is growing. The malignant cells consequently tend to spread from their focus of origin along the line of least resistance. Forcing their way along the lymphatic spaces they soon enter the small lymphatic vessels, pushing their way along these vessels in continuous lines. This route conducts them straight to the lymphatic plexus of the deep fascia. Thus in a very early stage of the growth an area of the deep fascial lymphatic plexus just beneath the primary growth is choked by the malignant cells. From this point the growth permeates the deep fascial lymphatic plexus in all directions, involving a continually increasing, but always roughly circular area of the plexus, an area whose central point is the primary growth. After a time cylinders of malignant cells will be pushed up towards the skin, or down into the muscles from this permeated area of the fascial plexus, but the infected area of fascia will always remain considerably larger than the infected area of overlying skin or underlying muscle. These facts give the key to the proper method of excising a melanotic sarcoma.

Embolic invasion of the lymph-glands.—The spread of permeation in the relatively small vessels of the fascial plexus is independent of the direction of the feeble lymph-stream. But when a cylinder of malignant cells intrudes into one of the trunk lymphatics which drain the plexus, the strong lymph-stream sweeps away small malignant emboli which lodge in the nearest set of lymphatic glands. These glands are invaded at quite an early stage, and consequently no operation for malignant melanoma is complete which does not include the excision of the regional lymph-glands. Indeed, if a malignant melanoma arises at the boundary line between two lymphatic areas, say, for example, in the middle line of the body, or along a horizontal line running through the umbilicus, it will be necessary to excise *two* sets of glands. Thus, if such a melanoma originates in the middle line above the umbilicus both sets of axillary glands would require excision. If it arose in the middle line below the umbilicus both sets of inguinal glands would require excision. If it commenced at the level of the umbilicus and upon the right side, the right axillary and the right inguinal glands would require removal. Moreover, permeation spreads around any and every focus of malignant melanoma, whether it be primary or secondary. Soon after the glands have been attacked by the emboli swept into them, permeation will begin to spread from the gland centrifugally in the surrounding area of the fascial lymphatic plexus. To meet this danger it is not sufficient simply to cut out the glands—a circular area of the surrounding deep fascia must be removed along with them.

The onset of blood-dissemination.—From what has been said it is clear that malignant melanoma is disseminated primarily by the lymphatics. Sooner or later, however, the slow processes of lymphatic permeation and lymphatic embolism are supplemented by blood-dissemination. And since the cells of a malignant melanoma appear often to survive when they get into the blood-stream—a point in which they differ strikingly from the cells of carcinoma—the slow processes of lymphatic dissemination are supplemented and practically replaced by the rapid and far-reaching effects of blood-dissemination.

Invasion of the blood-vessels from permeated lymphatics.—The manner in which this comes about is as follows: Along with the comitant vein or veins which generally accompany each small artery there generally run two comitant lymphatics. When permeation spreads along these lymphatics the malignant cells within the lymphatics, continuing to multiply, distend and finally split the lymphatics, just as ice bursts a water-pipe. The malignant cells are then free to multiply in the peri-venous and peri-arterial tissues. Infiltration of the thin wall of the vein follows, and the lumen of the vessel is finally invaded. A cylinder of malignant cells is soon found filling up the interior of the vein and pushing its way along towards the larger veins. When the larger veins are reached portions of the malignant mass are broken off by the force of the blood-stream, and are carried to lodge as emboli in the smaller pulmonary arteries.

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The embolised fragments grow into secondary pulmonary deposits, and soon afterwards, by processes similar to those already described, invade the systemic circulation. At this stage the whole body lies at the mercy of the growth, and the rapid formation of almost universal secondary deposits soon brings death to the patient.

Although the foregoing may be regarded as the typical course of events, it would appear that in some cases the cells of a malignant melanoma are unable to flourish in the blood. Under these circumstances the disease will run a chronic course, and, as generally happens in breast cancer, the fatal event is deferred until permeation has carried the cells of the growth along the lymphatics into the serous cavities. When this takes place widespread visceral dissemination occurs by means of "trans-cœlomic implantation," * a process I have described elsewhere; and the last stage of the case, though its onset is long deferred, is almost as rapid as in blood-dissemination.

The operation for melanotic sarcoma of the skin.—When malignant melanoma arises in the digits amputation should be performed at once. The operation should not be performed as if for a non-malignant condition. The flaps should never be cut so as to include any skin within, at least, an inch of the tumour. Moreover, they should be very thin, consisting only of skin and a very thin layer of subcutaneous fat, and not including the deep fascia. The flaps should be dissected up to a much greater extent than would be necessary merely to provide a covering for the stump. Over the whole of the area thus exposed the deeper subcutaneous fat and deep fascia should be dissected up towards the base of the finger or toe until the line of disarticulation or division of the bone is reached. The glands in the groin or the axilla are then to be removed.

When a melanotic growth occurs on the hand or the foot above the digits the question of amputation will always arise, but in early cases excision of the growth, even though it be attended by greater risk of recurrence, will usually be the operation of choice. If the glands are already palpably enlarged it is probable that anything short of amputation at the wrist or ankle is useless. When the growth starts higher up on the limbs, or on the head or trunk, amputation is replaced in most cases by excision. Museum specimens show how inadequate and empirical are the operations performed on these growths, even up to the present time. It is common to see the skin divided within a quarter of an inch of the edge of the growth. Invariably, so far as I know, the area of deep fascia removed is no larger than the area of skin excised, and consequently local recurrence in the neighbourhood of the scar is a very frequent event.

The principles upon which, in my opinion, the excision of a malignant melanoma should be carried out may be deduced from the mode of dissemination round a focus of melanotic growth. You will remember that there is permeation spreading centrifugally in the lymphatic plexus of the deep fascia, and invading skin and muscle secondarily over a smaller circular area. A circular incision should be made through the skin round the tumour at what is judged by present standards to be a safe and practicable distance. The incision, situated, as a rule, about an inch from the edge of the tumour, should be just deep enough to expose the subcutaneous fat. If necessary two radial linear incisions extending from the circular incision should be made on opposite sides of the tumour so as to facilitate the elevation of the skin flaps, which forms the next step. The skin, with a thin attached layer of subcutaneous fat, is now to be separated from the deeper structures for about two inches in all directions round the skin incision. At the extreme base of the elevated skin flaps a ring incision down to the muscles surrounds and isolates the area of deep fascia and overlying deeper subcutaneous fat to be removed. This fascial area is next to be dissected up centripetally from the muscles beneath up to a line which corresponds with that of the circular skin incision. Finally, the whole mass with the growth at its centre is removed by scooping out with a knife a circular area of the muscle immediately subjacent to the growth. The edges of the wound are to be brought together as convenience dictates. In every case the lymphatic glands which receive the lymph from the primary growth must be excised at the same time—a step the omission of which has often led to disaster.

In cases which show palpable enlargement of the lymphatic glands simple excision of the glands is likely to be quite useless. I have shown that permeation of the lymphatic plexus of the deep fascia soon takes place around the infected glands, just as it occurs around the primary tumour. The excision of the glands must therefore be carried out on exactly the same principles as the excision of the primary tumour, that is to say, a large circular area of the surrounding deep fascia must be exposed, dissected up from its circumference towards the infected glands, and removed in one piece with them. In late cases it may even be right to remove an area of skin over the infected glands, but such cases are probably inoperable.

* 'Cancer of the Breast and its Operative Treatment.' London, 1906, John Murray.

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There can be little doubt that the failure of surgery to deal successfully with malignant melanoma of the skin is partly due to causes within the surgeon's control, in other words, to defective methods of operation. The methods still employed in dealing with melanotic growths of the skin are precisely those which years ago gave such deplorably bad results in the treatment of carcinoma of the breast. Formerly the tumour, with a small circumferential area of skin, was cut out from the breast, and the axillary glands were removed, if at all, only when palpably enlarged. Even when the glands were excised the surrounding zone of permeated lymphatics in the breast, in the deep fascia, and in the muscles, was left intact to reproduce the disease. Nowadays the improved operation for breast cancer produces prolonged or permanent immunity in about 50 per cent. of cases, and there can be no doubt that the application of more thorough and scientific methods to the surgery of malignant melanoma of the skin will produce a corresponding improvement in the results of operation.

November 18th, 1907.

STONE IN THE KIDNEY AND URETER: SOME SIMPLE METHODS OF DIAGNOSIS.

By T. R. StJOHNSTON, M.R.C.S., L.R.C.P., M.S.A.,

Late Assistant Medical Superintendent Lewisham Infirmary; Late Clinical Assistant St. Peter's Hospital for Stone.

"Stone" is one of the ailments of mankind graced by the prestige of hoary antiquity; and, unlike some of the modern and more fashionable diseases, has been known and studied for so long that its symptoms have become quite classical; yet it is a condition that in its early stages often goes unrecognised, and even when advanced, if "colic" (or perhaps colic associated with but one other symptom) is the only evidence present, it may be too easily mistaken for some other disease.

Renal colic is so varied in character, and there are so many other things that will cause a somewhat similar pain, that the diagnosis of calculus often becomes a most difficult matter. Added to this is the fact that many of the associated symptoms—which ought to be confirmatory—are also found in other diseases of the urinary tract, so that instead of making matters more easy they only add to the confusion. Small wonder, then, that mistakes are often made. Let us take an imaginary case. After noting the patient's sex, age, general condition, and the history of any previous attacks, we investigate the very important symptom of *pain*.

Pain.—Asked what it is he complains of, the patient will probably say "attacks of pain in the loin, followed by a constant desire to pass water, which has a sediment, and sometimes a little blood in it." It is from symptoms such as these that we generally have to start our investigations. Now, the pain is the primary and practically the only constant feature. But "pain in the loin" is very vague. However, the character of it may help us a little. For most patients will agree that it is of a most terribly severe type, generally shooting downwards towards the testicle, and causing them to writhe in agony. Montaigne, the great essayist, in describing his own sensations has really drawn a picture of a very typical case. "Thou art seen to sweat with pain," he says: "to look pale, and red, to tremble, to vomit well nigh to blood, to suffer strange contortions and convulsions, by starts to let tears drop from thine eyes, to urine thick, black, and frightful water, or to have it suppressed by sharp and craggy stone that cruelly pricks and rends thee." Samuel Pepys, the old diarist, looked back with horror on the time when he suffered from "the stone." He used annually to celebrate the day on which he was relieved by operation, and such records as this continually occur: "March 22nd, 1661; this is my great day that three years ago I was cut of the stone, and blessed be God I do yet find myself very free from the pain."

But supposing the intense pain is the only symptom. How, then, are we to diagnose from those many acute abdominal lesions, not renal in origin, that start with a sudden attack of severe pain? To go into each one at length would fill too great a space, and their corroborative signs and symptoms will usually suffice to distinguish them. The question of sex, too, is a great aid. Ruptured tubal gestation or pyosalpinx are at once put out of court in the case of a male patient, as are also a ruptured or twisted ovarian tumour. In a woman a bi-manual examination will, of course, help us materially. But there are three great causes of severe abdominal pain which closely simulate renal colic and yet are not renal in origin. These

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are: (*a*) pressure from a neighbouring tumour; (*b*) lumbago; and (*c*) hysteria.

The commonest tumour in the region of the kidney is probably a lumbar abscess, due to tuberculous disease of the neighbouring vertebræ, but the actual pressure effects of this are seldom great, as being so soft and fluctuating it is readily adaptable to any cramped conditions of space.

In the case of a diseased vertebra, pain over the spinous process, rigidity of the column, and possibly an actual tumour will aid our diagnosis; but lumbago and hysteria, simple as they seem, will often require the greatest care in distinguishing from kidney trouble. In lumbago the pain is severe, sudden, and located over the region of the kidney; and hysteria, as we know to our cost, will simulate anything. In these two cases, then, we have to rely on other methods of diagnosis. Lumbago will cause none of the signs of constitutional disturbance usually associated with renal colic, such as pyrexia, vomiting, or rapid weak pulse; and in hysteria we may find an excessive flow of clear limpid urine (the reverse of that passed in renal colic), and the patient, usually a woman, may be taken off her guard and the pain disappear.

Turning now to those instances where pain is really due to some trouble in the kidney or ureter we cannot omit to mention such important things as movable kidney, tuberculous and malignant disease in the kidney or ureter, acute nephritis, and hydronephrosis. Movable kidney and floating kidney (with genuine meso-nephron) are really by no means such common occurrences as some physicians would make out, and when present are usually discovered in those who have lost much flesh, or in women after pregnancy, and usually on the right side. The pain is severe; indeed, Dietl, as far back as 1864, described "crises" producing all the pain symptoms of the worst renal colic, which he attributed to a temporary strangulation of the kidney or its vessels, but we can usually palpate a distinct and movable tumour, especially when the patient is examined standing; moreover, the other symptoms of a calculus are absent.

Tuberculous disease of the genito-urinary system is one of the greatest stumbling-blocks in our diagnosis of renal colic, since it furnishes so many symptoms closely parallel with those of the latter ailment. It may, of course, start primarily in the kidney, or in the epididymis or adjacent parts, but in any case the pain may be referred to the situation of the kidney. The character of the pain is usually dull and aching, but exacerbations often occur, and these, combined with the symptoms of frequency, pyuria, and possibly hæmaturia, render the diagnosis by no means an easy one. But in favour of tuberculosis we may mention that the pain is usually constant and not improved by rest, while that of calculus—when in the kidney—is relieved at night, and when the kidney is rendered comparatively immobile. The urine as an aid to diagnosis will be considered later.

Sarcoma and carcinoma of the kidney generally give rise to a continuous aching pain rather than the sudden anguish of renal colic, and a tumour can always be palpated. With carcinoma, too, a frequent sign is the presence of a varicocele, due to pressure on the spermatic veins. Hydronephrosis occasionally causes symptoms very comparable to those of renal colic, and the fact that a calculus itself may bring about a hydronephrosis, either by partially blocking the ureter or by the stenosis following injuries to the ureteric walls, should not be overlooked. But in simple cases of hydronephrosis the diagnosis is usually fairly easy, based upon a fluctuating tumour, which (if due to a kinking of the ureter) may at times disappear synchronously with the appearance of a large flow of clear urine. The only other important kidney lesion associated with severe pain is acute nephritis, when due to bacterial invasion, over-dosage with diuretics, certain drugs, such as cantharides and turpentine, or rarely, entozoa. The diagnosis of these forms of acute nephritis from renal colic rests mainly on the lack of other corroboratory signs of a calculus, and on the investigation of the actual cause of the nephritis.

The last reference to pain in its association with renal colic is the question of how it affects the patient. There are certain characteristics of the pain which are held to be strongly in favour of the presence of a calculus. The pain is usually described as a "tearing" pain, which shoots down the course of the genito-crural nerve, causing marked retraction of the testicle, owing to the spasm of the cremaster muscle; and there is sometimes even pain in the leg, inner side of the foot, and heel, due to the association of the genito-crural with the nerves supplying these regions. Guyon, of Paris, strongly defends the opinion

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that pain may be actually referred to the opposite kidney, which in itself may be perfectly healthy, but it is now generally considered that there really was some lesion in those cases where the other kidney has been cut down on and nothing found, although pain was associated with it. Speaking of referred pain, it must always be remembered that it may be referred *backwards* along the course of the nerves, and so a lesion in the bladder or genitals may cause pain over the region of the kidney. In cases of true ureteric pain the patient walks *with a lateral flexion of the body* due to his endeavour to relax the ilio-psoas and lumbar muscles, these being the ones in closest proximity to the ureter. And in conclusion it should be mentioned that the colic is due, not so much to actual laceration of the ureteric walls as to a stretching of them, and the spasmodic efforts of the ureter to contract and get rid of the offending body; it is well known that the rapid dilatation of any of the narrow tubes of the body is always associated with most intense pain.

Physical signs.—The prolonged examination of a patient during an attack of renal colic is neither justifiable nor satisfactory in its results, as the severe pain will cause so much rigidity that all proper palpation is discounted. But there are usually quiet intervals even when the stone is in the ureter, and at these times we may be able to extract certain information. It is said that in a very thin subject it is possible to feel a calculus in the ureter through the abdominal walls, though I must confess I have never yet succeeded in so doing; but an examination *per rectum* or *vaginam* will often definitely prove the presence of a calculus, and, indeed, one of the methods to force an impacted calculus into the bladder is by manipulation from the rectum. While making a physical examination it is as well to briefly note whether there is present any cause which may occasionally produce a hydronephrosis, such as a stricture in the urethra, or the presence of an enlarged prostate, with their consequently contracted bladder and the train of "back-pressure symptoms"; and also to examine the epididymis, vas, and seminal vesicles for possibilities of tubercle.

Urine.—The investigation of the urine is one of the most important aids to the diagnosis of calculus. The frequency of micturition should first be inquired into, both during the attack and in the intermittent periods. During the attack it assumes that condition known as "strangury," or the violent and constant attempts to micturate, with, however, only the passage of a few drops of urine each time. In the intermittent periods the patient will probably suffer from frequency in a modified degree, and then the diagnosis must be made from tubercle or any condition causing either cystitis or simple irritation of the neck of the bladder. The quantity of urine is important, as by this, combined with other signs, we are able to exclude such causes of vesical irritability as diabetes, and of pain as hydronephrosis or hysteria. While discussing the quantity passed, mention should be made of that interesting condition known as calculous anuria. It occasionally happens that a stone completely blocking the ureter will cause, not a hydronephrosis (which is a gradual occurrence), but a total arrest of secretion of urine—a "retention anuria," and in addition the other kidney will sympathetically also cease to secrete urine—a "reflex anuria." Such a condition is grave in the extreme, and calls for the immediate consideration of operative measures. In the case of simple renal colic the amount of urine passed is much diminished, but it must be remembered that the other kidney will often make up for this deficiency and bring the amount up to the normal standard.

The reaction of the urine is often looked upon as an indication of which portion of the urinary tract is affected, and also of the type of stone, if any, present; but chemical tests to differentiate calculi should not be relied on. There are several ways by which the character of a calculus may be ascertained, such as: (*a*) the pain is said to be more lancinating and acute when oxalic calculi are present, owing to their greater roughness and hardness causing more irritation to the ureter; (*b*) for a similar reason much hæmaturia and shreds of mucus are said to be indicative of oxalic rather than of other calculi; (*c*) the X rays also will show us a denser picture if an oxalic calculus is present than if a uric acid or a phosphatic one; (*d*) and lastly, the microscope will tell what crystals are present in the urine, though, of course, a mixed calculus may be present producing specimens of all the crystals. If the stone is in the mouth of the ureter or in the bladder the cystoscope will often solve the question of its character, and skilled manipulators of the sound can also differentiate the feeling of the several calculi.

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The question of hæmaturia is very important, and the diagnosis of renal calculus should never be decided till a thorough microscopical examination of the urine for blood corpuscles has been made. Often there is sufficient blood passed with the urine to apparently obviate the necessity of microscopical examination; but "we learn by our mistakes," and I shall never forget the diagnosis I made some years ago of renal colic in a patient who complained of sudden acute pain in the loin, accompanied by frequency and blood in his urine with no signs of tubercle. Fortunately a microscopical examination was made, and the ova of the *Bilharzia hæmatobia* were found, and it afterwards appeared that he had lately returned from South Africa, from a district where the bilharzia were rife, his pain being nothing but lumbago. Hæmaturia is sometimes present with tuberculosis, but is not very common.

Pyuria is found in many conditions of disease of the genito-urinary system, and its only significance in the question of renal calculus is that it indicates a chronic lesion, and that actual destruction of the kidney tissue is probably going on. The only satisfactory way to find whether the pyuria is renal or vesical is by examination with the cystoscope. The presence of pus in the urine may be overlooked in chemical tests, since the pus cells in strongly alkaline or ammoniacal urine are often completely disintegrated; but in all cases of pyuria it is advisable to make a bacteriological examination to ascertain whether it is tubercular in origin, and if so the bacilli are more easily found if the specimen of urine is previously digested for some twenty-four hours with a little pepsin.

Cystoscopy.—The last decade has brought about a small revolution in genito-urinary surgery with the advent of the cystoscope. No modern surgeon feels really safe in operating on a kidney unless he has the powerful corroboratory evidence of this instrument to tell him in which kidney the lesion lies. With it we can at once see lesions of the bladder itself, such as growths or tubercle, or a loose stone or one impacted in the ureteric orifice; and it is only when the stone is high up in the ureter, or possibly in the kidney itself, that any doubt may arise. To get a good cystoscopy it is essential to have the bladder well distended and to have a clear medium to see through. At St. Peter's Hospital the medium generally used is a weak boracic solution, and in all cases, except those in which hæmorrhage readily occurs, a clear fluid is a far better medium than an air distension, because there is practically no risk to the bladder wall from the over-heating of the cystoscope lamp; and, moreover, the character of the rhythmical whirls or spurts from the ureters can be better observed when they shoot into another fluid than when they simply pass into a bladder full of air. If a calculus is not visible it is from the ureteric orifices that we must draw our deductions, and we especially note two things, namely, the appearance of the opening and the character of the efflux. In the case of a calculus in the ureter itself, of recent date and with no accompanying pyuria, we may have one of two conditions. If the calculus has nearly reached the orifice there will be a slight dilatation of the mouth of the ureter, but the lips will not be thickened and outstanding as they are in the case of chronic pyonephrosis. But if the stone is higher up we may find a pin-hole contraction of the orifice, and this is due to a spasm of the ureteric muscle from the irritation of a rough calculus. When pyuria is present and the case is evidently chronic, possibly an old-standing calculus in the kidney, the orifice is retracted, the lips much thickened, and the space between them deep red in colour.

Looking at the efflux, the two sides are next compared, and it is noted whether both are normal in amount and rhythm, and whether clear urine issues from both of them. If the efflux on the suspected side, instead of coming out with a distinct swirl at set intervals, just trickles out of the opening, it denotes a loss of the valve-like action of the ureteric orifice, generally due to over-dilatation of the muscle walls by a calculus. There may, of course, be no efflux at all, should there be a complete obstruction; and, again, the efflux may be definitely blood-stained. Lastly, in the case of a pyuria where the pus is so small in amount that it cannot be distinguished from the urine in the efflux, one can catheterise each ureter in succession and find out conclusively which is the affected side.

X-ray examination.—The diagnosis of renal calculus has been materially aided in late years by the use of the Röntgen rays, but it is still difficult to always show the presence of a stone in a photograph. Mr. E. W. Shenton, Honorary Radiographer to St. Peter's Hospital, has recently published an article showing how much more satisfactory a screen view is than a negative, not only that it shows up the calculus better, but also that the respiratory movements affecting the kidney, which are the greatest drawback for a time-exposed negative, are actually of help in screen work, for in the case of a renal calculus they will show whether the suspected shadow moves *with* the kidney or keeps stationary while the kidney moves, therefore being unconnected with it.

Scybalæ are a source of frequent error, and it is advisable to take another view later on if a scybala is suspected, and order an enema in the meantime.

With the X rays we have, at present, come to the end of our scientific aids to the diagnosis of stone. What the future may bring forth remains to be seen; but the question of renal calculus is a difficult and serious problem, and any new method that will lighten the burden of doubt that even now so often assails us will receive the welcome that it will truly deserve.

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OBSERVATIONS ON SOME OF THE ACUTE PULMONARY DISORDERS IN CHILDHOOD AND INFANCY.

Delivered at University College Hospital.

By F. J. POYNTON, M.D., F.R.C.P.Lond.,

Assistant Physician to University College Hospital, and Physician to Out-patients at the Hospital for Sick Children, Great Ormond Street.

GENTLEMEN,—In this lecture my intention is to deal with some practical and clinical points in the more common and acute pulmonary lesions of childhood. I am led to the choice of this subject because there was an unusually severe visitation of virulent pulmonary infection during the earlier part of this year, and the experience that is gained from such an occurrence is always valuable and full of practical suggestions, and although these remarks must be desultory, they will be, I hope, useful.

These severe pulmonary lesions were associated with a recrudescence of influenza, and it is a dictum that pneumonia and pleurisy are sure to be more than usually fraught with danger when complicated by influenza. All experienced physicians directed attention to this fact in the earlier years of this present epidemic, and it is a menace to the young as well as to the adult.

It is under such conditions that creeping forms of pneumonia, foul empyemata, and gangrene of the lung are liable to occur, and the usual type of the symptoms in less severe cases is asthenic. Should measles break out, broncho-pneumonia, always a serious symptom in that disease, is more frequent and more severe—a fact which has even impressed itself upon our great public schools when nowadays so many boys are to be found who have never had the so-called children's ailments in their infancy.

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Pleurisy.

The first pulmonary condition to be considered is pleurisy. In young children and infants pleurisy is unquestionably a puzzling illness. The secret of this lies chiefly in the detrimental effect of *pain* upon such patients. Pain may kill an infant from the shock and suffering it entails, and yet the widespread fear of opium in the illnesses of childhood may lead to very imperfect attempts for its relief.

An infant, then, with pleurisy is very ill, and looks even worse than his actual condition is. There is a blue tinge to the face and lips, and an expression of much suffering, which, together with the sunken eyes, may lead to the natural suspicion that there is some deadly infection. The result of a physical examination, however, which is often a difficult matter, is singularly negative. For this, again, there is a clear explanation. You are well aware that it is a frequent occurrence to meet with pleurisy in an adult, without obvious pleural friction. Possibly, if you can persuade the patient to take a deep breath the friction may become apparent, but even then you may fail to detect it, although morally certain that there is pleuritis. No one, even if desirous to do so, can persuade an infant to take a deep breath when it has pleurisy, and so friction is often not detected even when it is present. A very significant sign, however, is distress on movement, for this entails some deepening of the shallow respirations and an immediate intensification of the pleuritic pain.

The air-entry may be impaired upon the affected side, but it has seemed to me that in young children the air-entry is often much impaired upon both sides. Even when it is unilateral, one is very liable to interpret it as evidence of a commencing pneumonia, because with pleurisy in the very young the temperature may be much raised, and pneumonia closely simulated. When the pleuritic pain is phrenic the distress is at its greatest, and the diagnosis often so difficult that it defeats the most astute, for pain is referred in childhood in an extraordinary way to parts far distant.

Among these cases occur some of the most striking instances of the simulation of *abdominal disease by thoracic disorders*.

The greatest pitfall is *appendicitis*. Appendicitis, as is well known, may occur synchronously with a right-sided pleurisy, but I want to bring to your notice also another accepted fact, that there may be pain and tenderness referred to the right iliac region, when the disease is confined to the chest. I do not wish to excite the opposition of those of you who are filled with the enthusiasm of surgery, but I would direct your attention to the exceedingly great difficulties that may surround the diagnosis of appendicitis, and ask you for this, among many reasons, not to accept the statement that all cases of appendicitis should be promptly operated upon without a thorough realisation of what such a rigid line of action entails. It takes for granted an ability to diagnose appendicitis which few of us possess. Anyone, of course, with a respectable medical education can detect the straightforward case, but it is the difficult ones which leave us in such doubt. You will all, I know, agree this far, that we ought not to operate upon the appendix when the condition is a severe pleurisy, yet a rigid rule of practice may lead us to do this, for the differential diagnosis is sometimes strangely difficult.

Another condition which phrenic pleurisy closely simulates in childhood is *general peritonitis*, for the upper half of the abdomen is immobile and tender, and often distended. The legs are drawn up, there is vomiting, and the facial expression anxious and sunken.

There can be no doubt that a cautious waiting policy is often of the utmost value in such grave and difficult cases, for pulmonary or pleuritic symptoms will certainly show themselves. Lastly, with pleurisy in infancy it is interesting to see the rapidity with which children come back from what appears to be death's door upon relief of the pain, for the cautious use of opium is the most effectual remedy.

Empyema.

Empyema in childhood is, all things considered, the most difficult of all pulmonary diseases.

Small empyemata are repeatedly found at post-mortem examinations which have not been diagnosed during life. On the other hand, a large empyema may be mistaken during life for a neoplasm bulging through the chest wall. It is always a disheartening thing to find an empyema after death, but it does not by any means follow that its discovery during life would have averted a fatal termination—a point to which I shall allude again later.

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A great step is made in the study of the condition when it is realised that it is not the pus of the empyema which constitutes the chief danger or gives rise to the most severe symptoms, but the infection producing the pus, for then you are freed from the rigid interpretation of such traditional and admittedly useful signs as irregular fever, wasting, sweating and anæmia. If the infection is virulent, the empyema recent and increasing, and the absorption from it rapid, such symptoms as the above will be present; but if the infection is not virulent, the pus slowly formed and shut off by thick layers of plastic exudation, there may be no fever, nor sweating, and comparatively slight constitutional disturbance. This chart, taken from a case of double empyema in an infant, illustrates this point decisively. On the other hand, acute tubercular pleurisy with a *clear* exudation may, on account of the virulence of the infection, be accompanied by sweating, high irregular fever, wasting and anæmia.

If a rigid interpretation of clinical symptoms is inadvisable an even worse mental attitude is that equally rigid one, which refuses "to guess" when it can explore and make sure. This is dangerous, because not even explorations can always make sure, and it is even more so, because it encourages a neglect of the habit of weighing evidence. With this neglect there supervenes a mental degradation which results in cases becoming doubtful that are in reality comparatively simple, and thus useless pain is inflicted and unnecessary anxiety caused to parents, who naturally fear explorations of the chest.

There are real dangers in exploration, and though exceptional they are thoroughly recognised. Pneumothorax is the most common. A more dangerous, and also direct, result is the unexpected coughing up of blood and death from pulmonary hæmorrhage—a disaster to which Dr. Carpenter has particularly directed attention. Œdema of the lungs and fatal syncope are also on record, and if aseptic measures are not rigid the lung or pleura may be secondarily infected. Without, I hope, burdening this point too much, let me ask you to picture your feelings, when, in a hasty moment, you have introduced a needle into the chest, the child has promptly coughed up blood and died of asphyxia, and the necropsy has shown a cirrhosis of the lung but no empyema.

A frequent occurrence in the empyemata met with in children is loud, whiffing, tubular breathing over the area. This is particularly liable to occur when the pus is unusually thick. I mention this point because loss of breath sounds is sometimes looked upon as a *sine quâ non* in the diagnosis of pleural effusion. Again, superficial medium-sized râles may be met with over the area of the lung in the vicinity of small empyemata. Much attention, then, should be directed to the intensity of the dulness and to any cardiac displacement, although it is my experience to find that when the latter is slight it is by no means easy to demonstrate with confidence.

Undoubtedly the great majority of empyemata are easy enough to detect by the usual routine examination coupled with exploration, and I need hardly add that in this country there are but few exceptions to the rule that for this successful treatment a portion of rib should be excised, although on the Continent it has been questioned.

Should an exploration find pus, it is advisable to operate over that point, and not to choose what might seem a more convenient site, viz. that of the usual seat of election. I have known that done and the empyema then missed because an adhesion had localised the abscess.

Occasionally you will be confronted with a case in which there is a large empyema of some duration with great cardiac displacement, and the child much enfeebled by the severity of the illness. Then I prefer a preliminary aspiration in order to allow the heart to recover a more natural position, my reason being the dangerous collapses that I have seen on the operating table when the chest has been dealt with in the usual way.

In a private house without all the perfect details and appliances of a hospital, this collapse may be fatal, and is inevitably most alarming.

Another point upon which I hold strong views is caution in shortening the drainage tube. I have seen most disastrous sequelæ from attempts at rapid closure. There may be a brilliant result now and then, but I am convinced that one or two such do not equalise the anxiety and pain which results when a small loculus of pus gets shut behind a too rapidly closing cavity. Many such failures have come to my notice. An oblique opening caused by inattention to the change in the position of the skin when the arm is raised at the time of opera-

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tion greatly increases the difficulty in the insertion of the tube subsequently.

Localised empyemata are the difficult ones. They may be found in almost any position, but there are three cardinal situations which are indicated in these diagrams and which are always well worth remembering: No. 1 is an empyema in the large fissure of the lung; No. 2 lies between the base of the lung and diaphragm; No. 3 abuts on the vertebral column.

It is in cases such as these that you will find your-

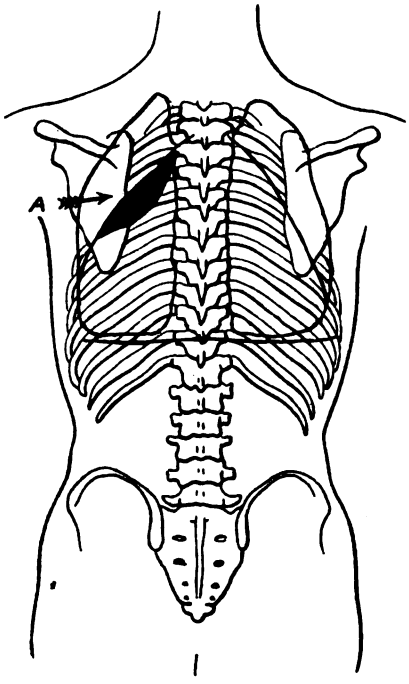

FIG. 1.—Diagram of an empyema in the large fissure of the lung. *A* is an exploring needle which has been inserted in the line of the abscess but has stopped short because frothy blood has been drawn from the overlying lung.

selves wondering, in the first place, whether you are dealing with an empyema, a localised, slowly resolving pneumonia, or a pneumonia with thickened pleura, or, again, tuberculosis of the lungs, or possibly a suppurative pericarditis. Then when you have decided that pus in the pleura is the most probable diagnosis, you will be hesitating where to explore, how far to thrust the needle, and how often to repeat the process. In hospital we are at a great advantage, because failures to detect an empyema are generally recognised as not unexpected, and the difficulties are thoroughly realised. Out of hospital, however, each failure is liable to be put down to lack of skill. One hopes that a gradual perfecting of the radiography of the chest will be able to afford us more assistance, but even then comparatively few of us will be able to obtain this help.

Unless the child is really too ill to risk an anæsthetic I greatly prefer some chloroform in these cases, and like to make up my mind first as to the most probable sites for exploration, and then in a leisurely way to introduce the needle at the different

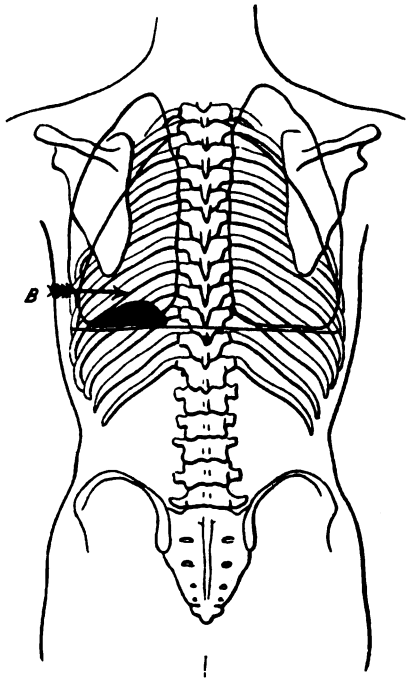

FIG. 2.—Diagram of an empyema between the base of the lung and diaphragm. *B* is an exploring needle inserted somewhat too high and passing directly into the lung.

points. Always test your syringe first, and on no account use too fine a needle, for then you may strike the abscess and yet get nothing. You will also realise from the diagram that you may be in the line of the abscess and yet be unfortunate enough to stop short, being arrested by overlying lung. An *intra-lobar empyema* is liable to burst into the air-passages, and in this way suffocation may occur. Should, however, this danger be escaped and pus be coughed up, the patient may make an uninterrupted recovery from that moment, and no hard and fast lines can be laid down as to the management of these cases. Each one must be carefully

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watched, and if the cavity is not being properly emptied a counter opening must be made and the abscess drained with this additional assistance. The position one adopts toward the complications of an empyema bursting into the bronchial tubes is thus clear. Prevent it if possible, for it may cause death by suffocation, but if it has occurred judge each case on its merits, and bear in mind that Nature may bring about as good a result as any operation.

Apical empyemata are rare. When they are upon *the left side* there is the natural fear that the

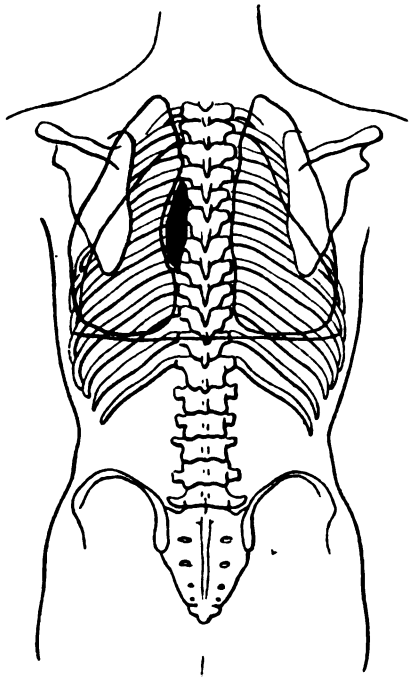

FIG. 3.—Diagram to illustrate an empyema between the vertebral column and lung.

dulness on percussion is due to some upward extension of the precordial area, and that exploration may injure the heart.

Suppurative pericarditis with a large effusion may indeed produce, as I have seen, absolute dulness up to the left clavicle, together with complete loss of breath sounds. In such cases the necropsy shows that this condition is partly the result of the collapse of the upper lobe of the left lung, which is pushed from the front of the chest by the encroaching pericardium, and partly due to the upward bulging of the pericardial effusion.

When an apical empyema occurs on *the right side* the heart does not cause the same difficulty in diagnosis, but there is one condition by which it is closely simulated, and that is tubercular infiltration of the upper lobe by a caseating bronchial gland. These infiltrations cause no shrinking of the lung, but may absolutely obliterate the air entry and give rise to stony dulness on percussion and high irregular fever. I have seen several explorations done, and have done the same myself under the impression that there must be pus. The diagnosis of a right apical empyema should be made with great reserve.

One of the chief dangers of an overlooked empyema is *suppurative pericarditis*. In former days, when operations were undertaken much more sparingly, this sequela was not uncommon. In searching through the post-mortem records of the Hospital for Sick Children, Great Ormond Street, for information upon suppurative pericarditis, I found that at least 60 per cent. were associated with a small empyema which had sometimes not been detected. Nevertheless it would be an error to conclude that all cases of suppurative pericarditis in association with empyemata have arisen as a direct consequence of the empyema, for there are virulent pneumococcal and other infections which attack many serous membranes simultaneously, and thus both pericardium and pleuræ may be independently infected. This is the point to which I referred in the earlier part of my lecture, where I stated that the discovery of an empyema at a necropsy did not necessarily prove that it was the immediate cause of death.

Suppurative pericarditis is a most deceptive condition, because pericardial friction is so often absent.

The most frequently recorded symptoms are wasting, pallor, irregular panting respiration, orthopnœa, vomiting, and fainting attacks. The temperature may be raised or subnormal.

I should not like to say how many times a small empyema with suppurative pericarditis has been mistaken for pulmonary tuberculosis, or to prophesy how many more times that diagnosis will be made. The blood count ought to help, one would think, but I am very distrustful of pinning my faith on the differential count of the number of leucocytes in a drop of a child's blood.

In all difficult cases of suspected empyema we should be on guard for pleuro-pericardial friction, for it may be the precursor of the true pericarditis,

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and points to the inflammatory process which is spreading from the empyema as having reached that part of the pleura which is adjacent to the pericardium.

Another danger that may result from a neglected empyema is *suppurative meningitis*. This is chiefly met with in marasmic infants, and may give rise to singularly few indications. The most suspicious signs are sudden unexplained vomiting and convulsions, with head retraction.

A third danger, fortunately rare in childhood, is *malignant endocarditis*.

Pneumonia.

It would be hopeless to attempt to enter upon the subject of pneumonia except in the most fragmentary way, but I should like to warn you against allowing parents to carry the process of hardening children to extreme. The general principle is well enough, but there can be no object gained by exposing young children to cold, damp, foggy air. Small children feel the cold intensely, and cannot express the fact that they are chilled. The parents are really at the mercy of the nurse, and she is often not really competent to judge and may be sometimes absolutely careless. I can give you two instances that occurred to me last spring.

In both cases the parents had very strong ideas upon the hardening of their children by regular open-air exercise; in both instances the weather was raw cold and foggy with easterly winds. Lastly, in both cases the children had slight colds, which were not deemed sufficient to keep them indoors. My contention is that in neither instance was the open-air exercise worth having, for one's only wish was to be indoors, warm and protected.

The results were appalling, for one of them was dead in three days from the worst broncho-pneumonia I have ever seen. The other recovered by what was practically a miracle after an illness, the gravity of which this chart will impress upon you.

The onset of pneumonia, though typically abrupt, is frequently in childhood preceded by indefinite illness, often gastro-intestinal, which may be protracted over some weeks. The pneumonia comes, as it were, with a final crash. I think this occurrence is a very puzzling one if you are not thoroughly acquainted with the fact. It is most commonly met with when there is an epidemic of influenza.

Apical pneumonia is a very interesting condition, more frequently met with in the young than in the old, and it may easily be overlooked. A very careful examination should be made above the clavicles and in the supra-spinous fossæ, and in one of these positions, and this only, may be discovered a most characteristic area of consolidated lung. The symptoms are often puzzling, for there may be very little cough, no expectoration, and severe cerebral manifestations, such as headache, vomiting, head retraction, stupor, and possibly convulsions. If, then, the pneumonia at the apex is missed, the diagnosis becomes most difficult and often impossible. It is the distinction between meningitis and apical pneumonia that is the great difficulty, and the rule—imperfect enough, it must be admitted—that seems to me to be most useful, is to withhold the diagnosis of the very fatal illness, meningitis, until there is some really definite evidence such as ptosis, squint, or other paralysis, or such ocular manifestations as choroidal tubercle and definite optic neuritis.

Sometimes a pneumonia runs its course for seven or more days without a definite physical sign of its existence, and then there comes a crisis and the appearance of consolidation. These cases are quite exceptional, but they almost always drive one to the error of calling them general tuberculosis and of giving a fatal prognosis.

Another interesting point is that among the cases of pneumonia this year there have been a good many in which a swinging, intermittent temperature has been a feature. One of the most common occurrences in this disease is to find a pre-critical fall of temperature followed the next day by a true crisis, but in these cases I am alluding to there may not be a sustained high temperature at all, or, again, after a few days of sustained high temperature there may be this phase of oscillations lasting a week or even longer. Each morning the child seems comparatively well; each evening he is flushed, his breathing is rapid, and the alæ nasi are working. Sometimes no evident cause can be demonstrated; at other times, small foci of fresh consolidation are detected.

When this period of oscillation is prolonged two difficulties are particularly liable to arise. One is a tactical difficulty. It is only natural that in a severe illness such as pneumonia we find that we are pressed by friends as to when the crisis will

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occur, and it is equally natural to give the approximate date. When, however, day after day the crisis appears to have occurred and yet the temperature flies up, the position becomes somewhat strained. I do not know any way by which this curious phenomenon can be foreseen, and can only say that when influenza is suspected no confident statements should be made upon the subject of the crisis in pneumonia.

The other difficulty is one of diagnosis, for the repeated rises of temperature undermine one's confidence, and the fears arise that the case is one of tuberculosis, or the chest is possibly explored for an empyema. These cases, however, seem to do very well; in due course the temperature swings less and less, and they progress to recovery without any bad symptom.

Earlier in the lecture empyema was considered from the point of view of pleurisy. If it is regarded from the point of view of pneumonia three types of onset are advantageously remembered.

No. 1. Empyemata which arise synchronously with pneumonia and sometimes pericarditis, meningitis, or peritonitis. These are never of large size, and are but one incident in a virulent generalised infection which proves fatal.

No. 2. Empyemata which supervene on a severe pneumonia, and hide the crisis of the pneumonia by maintaining the temperature at a high level. These are, in some respects, the most important class, because the dulness over the pneumonia changes almost imperceptibly into the dulness of the fluid of an empyema. If the gradual muffling of the breath-sounds and displacement of the heart are not appreciated, then it may be many days before it is realised that the condition is one of empyema rather than pneumonia.

The third type is the easy one, in which, after a pneumonia complicated by severe pleuritic pain has subsided, there follows an empyema, causing a rise of temperature with all the characteristics of fluid in the chest.

In some of the severe cases abdominal distension is a conspicuous feature. This, I have already mentioned, is sometimes associated with phrenic pleurisy, but this is not the case in the majority of instances. More usually this symptom develops towards the end of a severe case, when the strength is failing, undermined by toxæmia, fever, and respiratory distress. There may occur simultaneously with the distension an enteritis, or even a membranous colitis, with diarrhœa, blood, and mucus in the stools. Anæmic children are liable to develop gastric dilatation, and in exceptional instances acutely fatal distension of the stomach may supervene.

Two points of considerable practical importance arise out of the mention of these abdominal complications. One is caution in the pressing of liquid food upon children with severe pneumonia. As in typhoid the amount should be adjusted to the enfeebled state of the patient, and assistance be given to the weakened digestion by peptonisation, stimulants, and nerve tonics, such as strychnia. Children with severe pneumonia, I would add, do not, in my experience, bear large doses of strychnine, and are soon liable to show toxic symptoms, but small doses (half a minim for a child of two years) given every six hours are valuable.

Another useful remedy for abdominal distension is the old-fashioned asafœtida enema. It is not so irritating as turpentine, and often brings away much flatus and so relieves, for a short time at any rate, the cardiac and respiratory embarrassment, which is urgent when the upper segment of the abdomen is particularly implicated.

The other point is caution in giving aperients during the height of the illness, for an overdose may start a troublesome diarrhœa.

Sleeplessness is a serious matter in infancy, particularly if with it there is pain, and if, in addition, the child has been spoiled and there is struggling over the feeds. The strength is then soon exhausted.

Bromide of potassium, with a small dose of chloral (two grains for a child of two years), is often successful when insomnia is due to the fever and toxæmia. To this, nepenthe, half a minim, can be added if there is pain. Oxygen is also, in spite of adverse opinions, a most useful sedative.

Professor Northrup, of New York, has obtained great success with the open-air treatment of pneumonia. It is seldom that in a private house in this city there are conveniences for this, and our climate is damper and more treacherous. I have certainly no faith in doses of cold London fog. Warmed oxygen as an inhalation is infinitely preferable.

The helplessness of infants occasionally introduces curious difficulties, the mention of which

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may seem almost trivial. One of these interested me very much; the case was hanging between life and death, and the twelfth day had been reached without a crisis. The child was under two years of age, and that day he refused his food for the first time; the reason appeared to be that he was too breathless to swallow. A very excellent nurse pointed out that there had been some nasal catarrh, and that this, together with the fog—for the weather was very thick at the time—had plugged his nares far back with mucus and grime. The boy's only chance of breathing was accordingly through his mouth, and this was impossible when he was fed. The relief of this obstruction restored his power to take food, and the temperature touched normal on the fourteenth day.

On no account in desperate cases, when you thoroughly understand the condition, worry about examining the chest. You can do nothing. If the pneumonia is spreading it will probably mean death, and if it is not spreading the child wants all its strength for breathing and feeding. I once saw a marvellous recovery of a boy, who, so far as I could make out, had only the front of his right upper lobe left to breathe with. Splendid nursing saved him, together with the grand commonsense of his medical attendant, who just fed him and gave him a hypodermic of strychnine when collapse threatened.

Sponging, for example, often worries children, and should it do so it is far better stopped.

Pneumothorax.

The last condition to which I shall allude is pneumothorax. This is a very rare occurrence in children under twelve years. I can recall six under my own observation—two the result of exploration, one the result of a foreign body in the air-passages, two occurring in the course of acute tuberculosis, and one occurring in an infant with whooping-cough.

It may be useful to describe a case of pneumothorax occurring under observation. This patient was suffering from severe double pneumonia with pleurisy and double empyema. Eight ounces of pus had been withdrawn from the left side, and ten ounces from the right. In the evening after the second aspiration the boy suddenly sat up in great distress and said he was going to die. Examination found the heart beating just within the right nipple line and over the left lung there was hyper-resonance, a bell-note and metallic tinkling. It would seem that the pneumonic lung, softened by the disease, must have been injured either by the aspiration or the severe suppurative pleurisy, and have suddenly given way. Death ensued, but in this case not directly from the pneumothorax.

In other cases death has followed within three hours of exploration after a period of restlessness and distress with great dyspnœa, the respiration rate rising in some instances to 120 or even higher. Occasionally surgical emphysema in addition has immediately followed the operation.

Although a pneumothorax of tubercular origin is rare in childhood it does occur even in infancy as a complication of acute tuberculosis. In such cases a superficial cavity gives way as in the adults. Pyo-pneumothorax, too, may also follow in children with the classical signs of dulness at the base, hyper-resonance above and splashing sounds on shaking the chest. Death may also rapidly ensue after the lung has given way from disease just as it may in the traumatic cases.

These tubercular affections in infancy are rapidly fatal, and although a pneumothorax accelerates the end it is in reality but an unusal complication, and not a disease in itself.

In dealing with these cases each must be judged from its clinical characters. If the pneumothorax has, for example, followed an exploration with an aseptic needle, there is great hope that a pyo-pneumothorax will not follow, provided, of course, that there is not already an empyema upon that side. If the patient does not rapidly collapse and die and pus does not form, the other point to be remembered is the possibility of a valvular opening. If this is the case the air in the chest will collect under pressure, and the tension must be relieved by a Southey's tube, with trocar and cannula. The indications for this are increasing dyspnœa, increasing displacement of the heart, bulging of the intercostal spaces and enlargement of the affected side. The percussion note also becomes higher pitched and less drum-like.

When none of these events occur no interference is indicated, for the opening in the lung will be closed by inflammation and the air in the pleura gradually absorbed.

In conclusion, bear in mind that the bell-note may not be discovered even in the presence of an obvious pneumothorax.

November 25th, 1907.

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THE POSITION, DEFORMITIES, AND INJURIES ASSOCIATED WITH ADDUCTION OF THE FOOT.*

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Lecture I.

The deformities of the foot, except to a few enthusiasts, form a very uninteresting branch of surgery, their dull character being exaggerated by their representation in the text-books, as a series of distinct and separate conditions. It is otherwise in some works specially devoted to them. It is my intention to attempt to give a little life and interest to the subject by arranging them in two orderly series, into which they fall naturally, and which give the keys to their pathology and the explanations to much of their rational treatment.

The human foot has two extreme physiological positions, that of activity and that of rest or inactivity. In ordinary life it assumes a position which is a mean in relation to the extremes. These are positions or conditions of the foot which, it has been pointed out previously, contain within them the elements of grosser conditions, which we call deformities.† I hope, therefore, to give life and interest to these deformities by deriving them from the natural physiological conditions, thus bridging over the chasms which our ignorance and text-books have left between the natural and unnatural positions of the foot, and also between the different varieties of deformity. In the time at our disposal we cannot deal fully with so vast a subject and can only touch upon a few points. You must, therefore, excuse the ambulatory character of the lecture.

The deformities of the foot may be either congenital or acquired. The former are essentially "growth deformities." In their origin they are all similar. They are all due to the foot being retained in a certain position for a time sufficiently long to cause the constituent structures, bones, and soft parts to grow and adapt themselves to that position. For growing matter adapts itself to the physical conditions under which it grows, provided that those conditions are such as do not kill it.* The longer the growing foot is retained in the position the more permanent is the deformity. As the deformities were produced in this way, if we can reverse the conditions under which the structures grow we can reverse the process and correct the deformity. This is the object of massage and manipulations. It is the method by which they do good and can ensure cure. The process can be explained to the parents of the patient, who, at the same time, must be made to see that the treatment to be successful must be continued with unremitting care for a long time, one to two years. It is a striking fact that the treatment is far more successful amongst the better-to-do classes than amongst hospital patients, who often have neither the time nor the necessary knowledge to carry out the treatment fully and attain a success. In such cases it frequently behoves the surgeon to assist their efforts by some operation, which he is rarely, if ever, called upon to perform on the children of the better classes. †

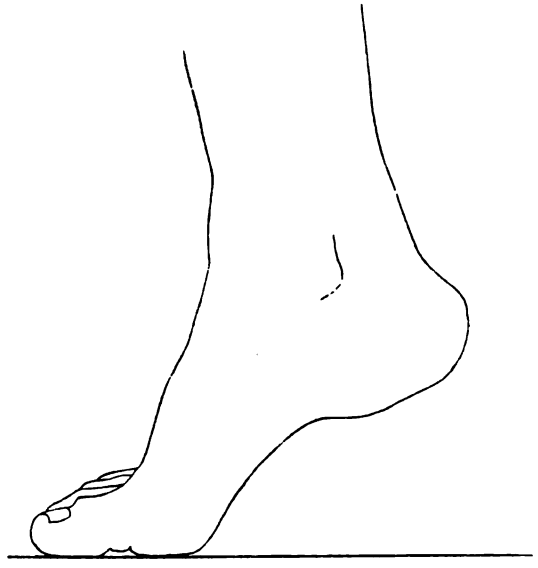

Fig. 1.—The foot in activity.

The deformities which interest us more in this

* Post-graduate lecture delivered at the Polyclinic and illustrated by lantern slides.

† 'Clinical Journal,' August 29th, 1906, pp. 309–315, and September 5th, 1906, pp. 328–334.

* Clifford Albutt's 'System of Medicine,' second edition, 1901, p. 1.

† "Common Causes of Failure in Surgical Procedures," 'Clinical Journal,' 1905, pp. 349–352.

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lecture are usually spoken of as acquired, not being obviously congenital in their origin. Some are acquired as the result of congenital conformations of the foot; others are acquired through accident or disease, such as the results of anterior poliomyelitis, infantile paralysis. As some are acquired from paralysis, which can affect any and various groups of muscles, it becomes incomprehensible to derive them from the active and resting positions of the foot, and in consequence I have substituted other names in the titles of my lectures—deformities associated with adduction and with abduction of the foot. Adduction is not a simple movement; almost all the tarsal joints participate in it. It is the position the foot assumes in activity and is associated with the deformities of talipes equinus, cavus, or varus, hallux varus, extensus or rigidus, digiti extensi. These deformities occur mainly at three joints; the equinus at the ankle joint, the cavus and varus at the mid-tarsal joint, and the remainder at the metatarso-phalangeal and interphalangeal joints.

The deformities associated with abduction, the position of the foot in rest, constitute the second lecture which I am to deliver to you. In this place it can only be stated that the various deformities, which are associated with it, occur at corresponding joints; the calcaneus at the ankle-joint, the planus and valgus mainly at the mid-tarsal joint, and the remainder at the metatarso-phalangeal and interphalangeal joints.

Before proceeding to the immediate subject I wish to point out a few various and easily recognised peculiarities by means of which people can be readily recognised to have adducted feet.

(1) Strong, active, and therefore usually young people commonly turn their toes in, or keep their feet straight forward; this is the conjunction of personal activity and inversion or adduction of the foot.

(2) Those who turn their toes in have arched insteps and adducted feet, and *vice versa*.

This demonstrates the conjunction of inversion with adduction and aching of the foot.

(3) The toes of the boots curl up to form a transverse crease across the "uppers" at the metatarso-phalangeal joints.

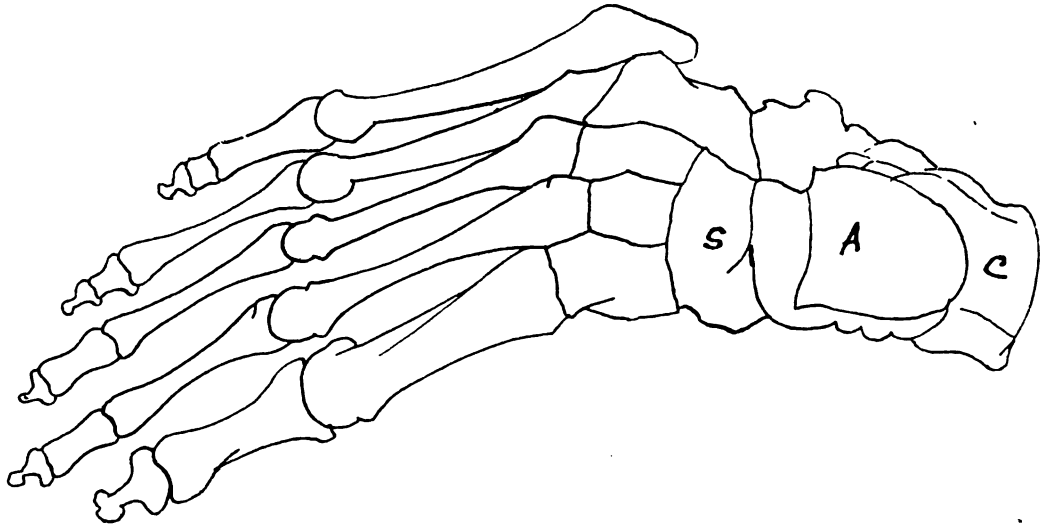

FIG. 2.—The bones of the adducted foot.

This demonstrates the extension (dorsal flexion) of the toes at the metatarso-phalangeal joints.

(4) The foot is short in an antero-posterior direction, thick from above downwards.

(5) The heel of the boot is worn approximately in the middle line and the tread is mainly on the inner side.

(6) The callosities on the sole of the foot are on the back of the heel, slightly to the outer side, and under the big toe and extending outward.

(7) These people are not always steady when standing. It is the position of the quick and alert, not being meant for steadiness but activity.

(8) The deformities associated with the position of adduction are:

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(*a*) A slight degree of equinus, sometimes spoken of as right-angle talipes.

(*b*) Inversion of the foot, talipes varus.

(*c*) A high arch, talipes cavus.

(*d*) Hallux extensus and its rigidus.

(*e*) Hallux varus, inversion of the big toe.

(*f*) Hammer-toe.

(*g*) Digiti extensi, the toes being extended at the metatarso-phalangeal and flexed at the interphalangeal joints. Hallux extensus and hammer-toe are merely specially recognised and named examples, like the digitus minimus extensus mentioned in the sequel to these lectures on the results of high heels.

Only a few of these conditions merit special mention, and such as do will be discussed *seriatim*.

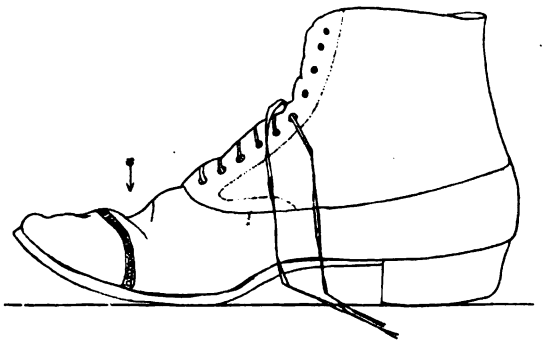

FIG. 3.—The boot of a patient with pes cavus, showing the curling up of the toe and the transverse creasing.

Talipes equinus.—Of this deformity there exist very many varieties which in their various degrees are so well known that it is unnecessary for us to go into them. But there is one variety, which is commonly overlooked, to which it is important that attention should be directed. In these cases the foot can only be extended (dorsally flexed) to form a right angle or a little more or less with the leg. As in walking the foot is placed on the ground in front of the body, which then swings over it, the ankle-joint being at first flexed (plantar flexed) and then extended (dorsally flexed). If the latter movement is limited, as it is in the cases of right-angle talipes under consideration, the patient cannot walk well unless steps are taken sufficiently short to suit the limited movement at the ankle-joint. Thus to many people this limitation of extension (dorsal flexion) is no great disadvantage. Take, for instance, the short steps of women in whom this limitation is common, the limitation being often acquired from the habit of always wearing high heeled shoes.

But in more severe cases, besides the shortness of the step, the patients suffer from pain, and perhaps corns, under the ball of the big toe. Or they may complain of pain in the tarsus, such as might suggest the possibility of tuberculous disease. Indeed, I know of one case in which the tarsus was trephined, the pain in reality being due to the pes cavus. A combination of these two factors may absolutely incapacitate a patient from walking. Besides the right-angle talipes, these patients have high insteps and toes which are curled backwards; hallux extensus, hammer-toe and digiti extensi. And the practitioner can be easily misled by the pain complained of in the ball of the great toe, the hammer-toe, or the pain in the tarsus, in any of which cases his treatment will not cure his patient, and perhaps not even give temporary benefit, because the primary lesion is situated above the sites of these symptoms. It is in the ankle, and no modifications of boots or corn plasters will cure it. The first point to decide is whether it is right in a given case to attack the trouble at its source or to be content with the amelioration of the special symptoms. A pad under the arch will do much to relieve the pain under the big toe and in the arch of the foot. A bed can be made in the sole for the ball of the great toe. A hammer-toe can be treated by operation. Bars, similar to those on football boots, across the sole of the boot often relieve the pain. But none will cure the limitation of movement at the ankle-joint. If it is decided to attack this, the question must be decided whether the tendo Achillis is at fault or the movement is limited by the anterior margin of the lower end of the tibia coming in contact with the astragalus. The practice of blindly dividing the tendo Achillis without sufficient forethought is not infrequently disappointing in these cases, and the reason why is not far to seek. The high arch, so much favoured by mothers when in their children, brings the astragalus prematurely in contact with the anterior edge of the lower end of the tibia in extension. It may be necessary to do something to both bones. This is well shown by the astragalus of a sailor. But prevention is infinitely better than cure, and the ankles of growing children should be educated like

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the rest of their bodies and brains. If neglected in childhood, the condition causes more discontentment than actual disaster, because it makes the possessor a second- or third-rate athlete in all senses of the word. Yet the discomfort produced is not enough to make it justifiable to advise operative relief. Moreover, if his ankles have been neglected so will, in all probability, the much more important athletic factor—his chest.* The time has gone by to make an athlete when growth has ceased.

Pes cavus.—In another place † I have shown that a high arch is a symptom of the inversion or varus which accompanies the greater degrees of adduction of the foot. Previously it had been supposed to have been dependent on the accompanying degree of equinus, but this is not so. It results from the physiological correction of the varus, as in standing or walking. Thus the presence of a high arch or pes cavus is not a disease or deformity in itself, but a symptom of the adducted position of the foot. An arch is always to be seen in an active or athletic foot. The symptoms which may arise from pes cavus are very like those just narrated under talipes equinus, the similarity emphasising the sisterhood of the conditions, pain in the tarsus, pain under the great toe, and, perhaps, hammer-toe. These can be met as already described; but the questions must be considered as to whether the ankle-joint was or not primarily at fault, and if any treatment should be directed to it. Thus it can be seen that deformities of the feet require very nice consideration and decision.

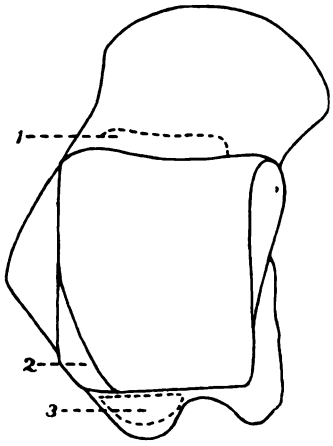

FIG. 4.—Astragalus of a sailor, showing additional facets (after Mr. Arbuthnot Lane). 2 articulates normally with the fibula; 1 and 3 are the additional facets for articulation with the tibia, which have been developed in consequence of the character of his work as a sailor.

But all forms of pes cavus are not of this type, and there is one which I would specially commend to your attention as it offers the opportunity of tendon grafting, which gives better results than almost any other.

Regarded from the point of view of treatment, the most promising case is where the paralysis affects the peronei muscles only, or perhaps also the anterior tibial group only slightly. The peronei being out of action, there is no opponent to the action of the tibialis posticus muscle. As the tibialis posticus is a muscle to which but little attention is directed by anatomists and the action of which few know, I shall be excused if I quote a short passage. "The tibialis posticus is able and does perform alone the movement of adduction of the foot, and that all the other muscles are engaged simply in retaining the equilibrium, or balance at the several joints." As I have already said, and shown elsewhere, an increase of the arch of the foot is an integral part of the movement of adduction. So that with paralysis of the peronei the unantagonised action of the tibialis posticus is seen clinically as a paralytic pes cavus; or another name has been suggested for the same condition, pes adductus. Provided that there is not much paralysis in the anterior tibial group of muscles, by the operation of tendon grafting a great deal can be done to benefit these cases, the rationale of which I can briefly indicate and commend to your notice. The tendons of the peronei and the tendo Achillis are exposed through a flap incision. The latter tendon is split and the outer half cut across transversely close to the os calcis. The peronei tendons are punctured in three places and the separated portion of the tendo Achillis threaded through them, its end being fixed by a single stitch at the third puncture. There is no need to use

* Clifford Allbutt's 'System of Medicine,' second edition, vol. i, article "Physical Exercise," pp. 382–421. 'The Archives of the Roentgen Ray,' "The Importance of the Development of the Chest in the Young," March, 1905, vol. lvi, pp. 219–223. 'The Archives of the Roentgen Ray,' "The Physiology of the Chest in Exercise," 1905, vol. lxiii, pp. 133–135.

† 'CLINICAL JOURNAL,' August 29th, 1906, pp. 309–315, particularly 312–313.

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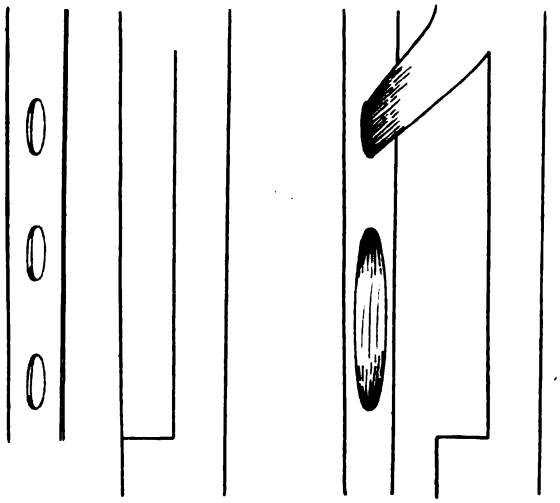
the fingers in this operation, which, to ensure success, must heal by first intention. Movements are begun by the patient within a few days. After the operation the foot is firm on the leg, whilst previously it "wobbled about" when the leg was shaken from side to side. By means of threading part of the tendo Achillis through the peronei two advantages are gained. Firstly, the threaded tendons can never separate; secondly, to do so necessitates that the tendo Achillis is split right up to the muscles attached to it, so that the outer muscles, *e.g.* the external head of the gastrocnemius, act on the peronei tendons, and to a great



I. II.

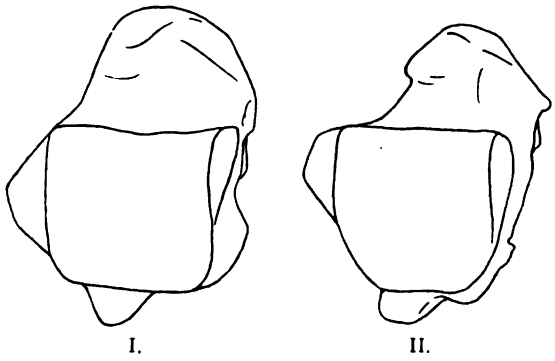
FIG. 5.—I. The peronei tendons on the left have been pierced in three places, and the tendo Achillis has been split and the part next the peronei detached. II. The detached portion of the tendo Achillis has been threaded through the peronei tendons.

extent, having separate nerves from the inner muscles, the patient has a better chance of learning to dissociate the actions of the two groups.

To attain the maximum of possible success it is often advisable in male patients to perform later a tenotomy of the undivided part of the tendo Achillis to correct the small degree of equinus so often found in these cases, by these means giving free movement of extension at the ankle-joint. In female patients it is usually not worth while to do so on account of the high heels which they will wear.

Further, to enable the arch of the foot to be lowered, it may be desirable to lengthen or divide the contracted tendon of the tibialis posticus.

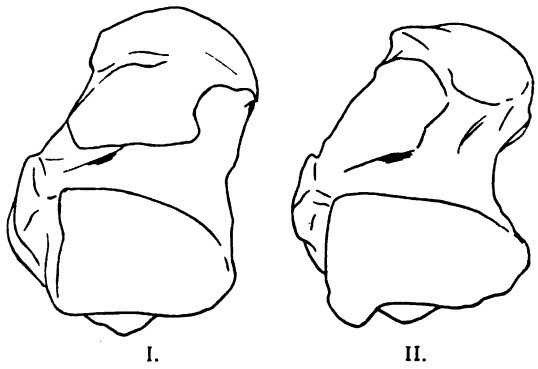
Talipes varus is the inversion of the foot which accompanies its adduction, the unfortunate possessor walking on its outer side. In the slighter degrees it is easily corrected by teaching the child to walk on the soles of its feet. In later life, whenever necessary, it can be treated with advantage by excision of the astragalus. The specimen



I. II.

FIG. 6.—I. A normal astragalus. II. An astragalus removed from the foot of a woman affected with infantile paralysis.

shown was removed from a woman, æt. 40 years, who had a paralytic club foot. Before the operation she walked on the outer side of the foot, and was quite content until she suffered pain. The



I. II.

FIG. 7.—Under surfaces of the same astragali, showing the deformity of the articular surfaces between the astragalus and calcaneus produced by adduction of the foot. I. Normal astragalus. II. Abnormal astragalus.

astragalus, shown you, was removed through an external incision. It shows well the inward bending of the neck which is a deformity resulting from the growth of bones subsequent to the incidence of the infantile paralysis. This and the poor development of the bone from the paralysed limb are the two most conspicuous features to be noticed.

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Hallux varus is an inverted condition of the great toe; it is seen as part and parcel of talipes varus and occasionally as a congenital deformity.

If one throws one's foot into a position of strained activity with the heel raised, the toes are extended (dorsally flexed) at the metatarso-phalangeal joints, flexed (plantar) at the proximal interphalangeal and extended at the distal interphalangeal joint, a position called hallux extensus in the great toe, hammer-toe in the second toe, and digiti extensi in general.

In the normal metatarso-phalangeal joint of the great toe the proximal phalanx moves round the head of the metatarsal on the arc of a circle whose radius is the lateral ligaments. With the adducted foot and its raised arch much weight is thrown on the ball of the big toe, sometimes making it so painful as to cripple the patient. In consequence the head of the metatarsal becomes enlarged on its plantar surface, in a way similar to that of any bony part exposed to intermittent pressure, such as the tip of the os calcis in the heel. In consequence of this, the proximal phalanx can no longer be flexed plantarwards, the tight lateral ligaments forbidding it to ride over the bony enlargement. The toe may yet remain more or less straight and the condition has been called the *hallux rigidus associated with the hallux extensus of the adducted foot.* It is the precise reverse of the hallux rigidus associated with the hallux flexus of the abducted foot. As the enlargement on the under surface of the head of the metatarsal increases in size, the toe is slowly pushed more and more upward into the position of *hallux extensus.* Palliative treatment is usually all that is required, such as suitable boots, a pad under the arch, and a bed made in the sole for the ball of the great toe. If operative treatment is required it usually consists of amputation at the metatarso-phalangeal joint.

Besides these deformities, one, digitus extensus of the great toe, has been reserved for consideration in the second lecture; and another, minimus digitus extensus, to a sequel to these lectures on the consequences of wearing high-heeled foot gear.

In severe cases, the toes, *digiti extensi*, are drawn backwards on to the dorsal surface of the metatarsal bones and can no longer touch the ground. Under which circumstances, if they are troublesome they had better be removed as they have become useless, the heads of the metatarsals projecting beyond the toes, which are even dislocated on to the dorsum of the metatarsals.

Lastly, we come to the consideration of *hammer-toe* (an example of digitus extensus), one of the most frequent of the deformities of the active or adducted foot to demand special treatment. The deformity attracts attention to itself in the second toe on account of its greater length and prominence. So that when it is doubled up it rubs on the upper and the sole of the boot. Splints and such treatment can at best only attain very partial success, because the hammer-toe is a deformity which is merely a symptom of the adducted foot. So that so long as the foot remains adducted the hammer-toe (digitus extensus) must recur. If operative treatment is demanded amputation is the most logical, but has been advised against on account of the bad hallux valgus which may follow. But this post-operative mishap has certainly been much exaggerated. A bent up second toe, such as it is before operation, can have little or no influence in preventing the development of hallux valgus. Certainly amputation is the best treat-

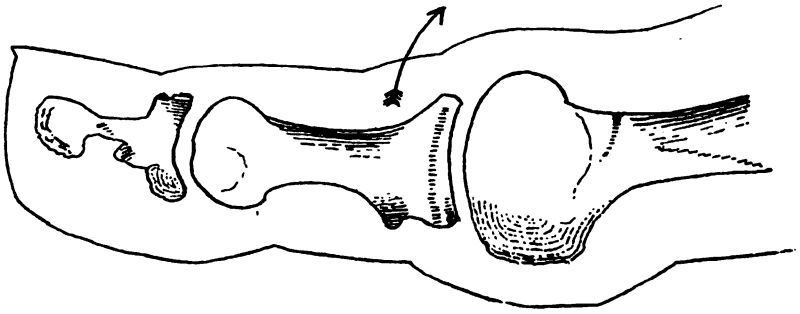

FIG. 8.—The hallux rigidus associated with hallux extensus. Compare with Fig. 4, Lecture II, in which the deformity of the metatarsal is on the dorsum, whilst here it is on the under part.

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ment in ladies and most women who wear shoes with pointed toes. The method of excising the head of the proximal phalanx is a half measure, often successful because it so shortens the toe that it no longer rubs against the upper of the boot. But the "hammer" deformity will surely recur. It is unwise to try and make a stiff toe by excising the proximal interphalangeal joint and obtaining bony union between the fragments. This operation, in fact, has owed its success to the failure to secure osseous union. Tenotomies of the tendons are unlikely to be successful and should not be employed.*

A deformity at the interphalangeal joint of the great toe associated with the position of adduction will be considered in the second lecture along with the corresponding deformity of the position of abduction. The latter deformity has been called *hallux retractus*.

Associated deformities.—Besides those of the foot itself, other deformities are often associated with the adducted position. For instance, the ankles tend to bend outward. And it is a most striking example of favourable adaptation that Nature has prolonged the fibula into a process of bone on the outer side of the joint, the external malleolus, to prevent this tendency to an outward displacement, associated with the active and adducted position of the foot. No such provision has been made with the internal malleolus to prevent the in-bending of the ankle, because it is associated only with the inactive resting position of the foot. It was unnecessary in people whose existence depended on their activity. The outward bend of the ankle is transmitted to the knees, which bend outward—genu varum or bow leg. And it is a common observation that men with slight bow leg are always strong and active. In fact, a slight outward bow in the lower third of the tibia is frequently seen in athletes or observed in *premiere danseuse*. The femur in these subjects is more curved than in others; the spine and trunk generally are extended and in the position of activity.

Injuries peculiar to the adducted foot.—The injuries peculiar to the adducted foot will only make themselves apparent when the patient falls squarely on the sole of the foot. The force is distributed on the heads of the first two metatarsal bones, the base of the fifth, and the heel of the os calcis. Fractures may occur in any of these situations or in the astragalus as the result of the tibia being forcibly driven on it. The tendency of fractures in this situation is to make their presence felt by pain rather than absolute disability. For instance, a young engine driver with an adducted foot fell from the top of the cab of his engine, about fifteen feet, on to his foot. After six weeks or two months he was not well and was sent to me at St. Thomas's Hospital by his doctor, Dr. Mathews, of New Cross, for an opinion. He had been skiagraphed at a large London hospital shortly after the accident when nothing was seen. Yet the man had a very constant line of tenderness, easily demonstrable, on the outer side of his os calcis. Mr. A. H. Greg, the radiographer to St. Thomas's Hospital, was able to show the existence of a fracture through the os calcis is this situation; union had not yet

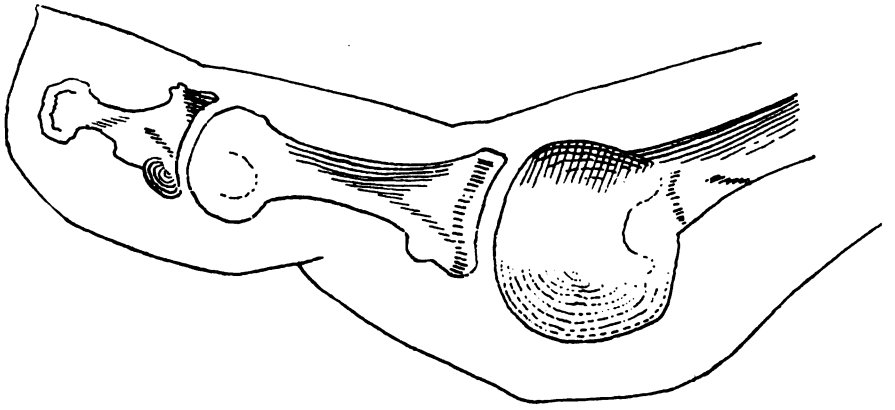

FIG. 9.—Hallux extensus with enlargement of under part of the head of the metatarsal.

* 'Operations of General Practice,' The Oxford Medical Press, pp. 247-249.

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taken place, doubtless due to the movements of the man's attempts to stand and walk.

A slip or slight fall, with the adducted foot slightly inverted, is quite sufficient to fracture the base of the fifth metatarsal bone, the presence of which will give rise to pain for some weeks or months. There will be a constant tender spot, in

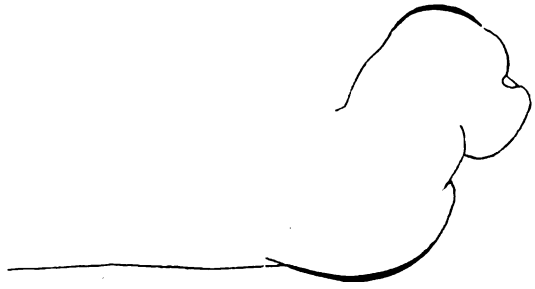

FIG. 10.—Retracted little toe, formed with adducted foot, digitus minimus extensus. It does not touch the ground.

all probability no crepitus will be found, and a skiagraph will demonstrate the fracture. The first metatarsal bone rarely breaks.

In fact one cannot too strongly advise that good skiagraphs be taken as a routine practice in all cases of foot injury, particularly those of apparently slight injury which do not get well within a reasonable time.

November 25th, 1907.

DISEASES WITH DROPSY AND MUCH ALBUMEN.

BELONGING to this group are acute Bright's disease (acute parenchymatous nephritis) and the various forms of chronic parenchymatous nephritis, as fatty kidney, large white kidney, waxy kidney, and the contracted form of the white kidney.

The diagnosis of acute nephritis is made on the following facts: A history of an exciting cause, as scarlet fever, diphtheria, and exposure to cold; often, although not always, œdema beginning in the face, especially about the eyes, and spreading to the limbs and trunk. There is a fairly sudden onset, scanty urine, which contains much albumen and blood, has a high specific gravity, and holds numerous epithelial and granular casts. The heart is not hypertrophied unless it be an acute exacerbation of a chronic diffuse nephritis. In febrile albuminuria the amount of albumen is slight, and there are no blood-corpuscles or epithelial casts.

The diagnosis of chronic parenchymatous nephritis, large white kidney (which may follow an acute nephritis, but more often not), is made on the pallor and often the dropsy of the patient (giving the "large white" body), an amount of urine somewhat below the average of 40 oz., a specific gravity somewhat raised, a considerable amount of albumen, and large numbers of fatty and epithelial casts. The heart is hypertrophied. The duration of the case is probably about two to six or seven years. There is often albuminuric retinitis and uræmia.

Amyloid kidney presents much the same clinical features, but there is a concomitant cause of lardaceous disease (syphilis, chronic suppuration), and there may be enlargement of the liver and spleen with persistent diarrhœa. The amount of urine is variable, but as a rule there is early polyuria; the specific gravity is also variable, the amount of albumen is large, and there are comparatively few casts, which are mostly hyaline or waxy. The heart is not hypertrophied, and there is rarely uræmia or neuro-retinitis.

In the secondary contracted kidney there is a history of long-standing kidney disease, a year and a half to three years, with clinical signs of the earlier stages, passing into those resembling cirrhosis—namely, much urine, of a low specific gravity, slight albuminuria, and few casts with a hypertrophied heart. But the amount of urine is less than in interstitial nephritis, the albumen more, and the casts are epithelial, not merely hyaline, as in that disease.

The above is taken from Dr. Arthur Whiting's book entitled 'Aids to Medical Diagnosis,' published by Baillière, Tindall and Cox. The author, in his preface, draws special attention to the fact that the basis of the work is clinical and not pathological. The book is, therefore, of assistance in cultivating the power of diagnosis. There is an excellent section on differential diagnosis of infectious diseases which has been contributed by Dr. Herbert Cuff, for many years in charge of one of the larger fever hospitals under the Metropolitan Asylums Board. Dr. Whiting has certainly succeeded in producing an excellent volume for the purpose for which it was intended, and not only advanced medical students but also post-graduates will find it useful and readable.

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THE CLINICAL JOURNAL,

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON

CIRRHOSIS OF THE LIVER IN CHILDREN.

Delivered at University College Hospital.

By C. BOLTON, M.D., B.Sc., M.R.C.P.,

Assistant Physician to the Hospital.

GENTLEMEN,—Cirrhosis of the liver in children is much less common than it is in adults, but still it is by no means rare. The absolute frequency of the disease in children has been worked out in different countries. In England it occurs in about ·2 per cent. of autopsies in children under twelve years of age. In other countries it varies somewhat. England is, of course, noted for its frequency in alcoholic cirrhosis. A very great point of interest in cirrhosis in children is the diagnosis, because there are very many diseases which are liable to simulate cirrhosis clinically. This little boy was brought up to my out-patients at the North-Eastern Hospital for Children on January 8th of this year. I will first give you the notes of the case, and afterwards we will discuss the different varieties of cirrhosis found in children. His age is 7 years, and he was brought up because he had jaundice. The present illness dated from a few weeks before admission. He had been losing flesh for this time and the jaundice commenced four days before admission. The motions were light in colour, and he had itching of the skin, and some diarrhœa. There was headache and drowsiness but he never had any bleeding of the nose, melæna, or hæmatemesis. He had vomited once, but it was after taking medicine.

With regard to past illnesses, he was born at full term and had measles when a baby, and whooping-cough when he was three and a half years of

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age. There was no history of congenital syphilis, and no history of alcohol nor of scarlet fever. The family history was very good; there was no history of syphilis, and the father and mother were perfectly healthy. His mother had had two miscarriages, and the boy has one sister who is eleven years of age, quite healthy. Three children died of broncho-pneumonia. No other disease of this description has been seen in any member of the family. On admission he was found to be well built and looked his age, although he was rather thin. There was no clubbing of the fingers. He had deep jaundice. The urine contained bile pigment, and the pathologist sent a report stating that bile salts were absent. The stools were pale. His appetite was good, and there was no pain, diarrhœa, nor any other symptom whatever. His teeth were a little carious and the tongue slightly furred. On examining the liver it was found to be enlarged downwards; the upper limit was at the normal position, and the lower margin extended to a point about midway between the ensiform cartilage and the umbilicus. It was firm and the edge well defined. Projecting below the lower margin of the liver was a large globular mass in the position of the gall-bladder. It seemed too large and too hard to be the gall-bladder however. It was obviously attached to the liver and moved with respiration. The spleen was just felt below the costal margin and there was no free fluid in the abdomen. The heart and lungs were normal and the temperature on one or two occasions during his stay in the hospital was raised for a few days, but it reached no higher point than 102·8° F. I thought the case was probably one of tumour of the liver pressing on the common bile-duct and giving rise to jaundice. I decided to have the case operated upon, and therefore called in my friend Mr. Stabb. He opened the abdomen on the right side, and on examination found that the globular mass was a part of the right lobe of the liver itself, projecting beneath the edge. It was a very remarkable condition. The spleen was found to be considerably enlarged, and the pancreas normal. There was no tumour nor enlarged glands to be felt, and there was no ascites. I asked Mr. Stabb to remove a small piece of this globular portion of the liver for examination, and I have put a section under the microscope for your inspection It proved to be a case of cirrhosis. The capsule was a little thickened, and the fibrous change not very extensive. The fibrous tissue is present between the lobules, and in places it extends into the lobules leading to destruction of the liver-cells. The liver-cells are separated from each other in places, and islands have been formed. No new bile-ducts can be seen at all. The origin of this change is probably as follows: There has been an infection through the bile-ducts, and this has led to cirrhosis around the bile-ducts, so-called biliary cirrhosis. I think it is an early example of Hanot's cirrhosis of the liver. After this the patient made an uneventful recovery, and, as you see, he is quite well, the jaundice has all disappeared, but you can still feel the liver, and I shall ask you after the lecture to examine him. This case belongs to a clinical type of cirrhosis in children, in which there are enlargement of the liver and spleen, jaundice, ascites, and toxic symptoms. At one end of the scale there is Hanot's cirrhosis, in which jaundice is very marked and ascites is practically absent, or only comes on towards the end of life. At the other end of the scale there is multilobular cirrhosis, atrophic or alcoholic cirrhosis, in which ascites is a marked symptom and jaundice is commonly absent, or if it is present it is only to a very slight extent. Quite distinct from this group of cases there is another class in which ascites is the chief symptom, practically the only symptom of disease of the liver. Thirdly, there is yet another class in which there are no direct symptoms referable to liver disease; one simply finds enlargement of liver, and attention has been drawn to the fact that the liver is affected by some general disease that the patient is suffering from in which an enlarged liver is likely to be found. So that there are three distinct clinical types of cirrhosis in children. Type I, in which there are direct liver symptoms present, and of this there are two classes: (*a*) conditions like Hanot's cirrhosis, and (*b*) conditions like multilobular cirrhosis. Type II, in which ascites is the main symptom, jaundice and toxic symptoms being absent. Type III, in which the only sign is enlargement of liver, which can be felt on palpation.

I will first take up Type I, describing to you the different forms of cirrhosis met with. *First, Hanot's cirrhosis* (hypertrophic or unilobular cirrhosis). The symptoms of this condition are prac-

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tically the same as occur in the adult, so that I need not go into them very particularly. One sees marked and early jaundice; the liver and spleen are both enlarged; in some cases the liver is larger in proportion than the spleen, and in other cases the spleen is much more enlarged than the liver. Ascites, if it is present, is not very marked. Hæmorrhages from the gums, nose, or stomach may occur, and death from coma is very common. It runs a fairly chronic course, sometimes lasting four or six years. If one meets with a case of this description, one diagnoses Hanot's cirrhosis if no ætiological factor such as alcohol or acute specific fevers can be found. Clinically, the case which we have before us appears to me to be one of this type, and is apparently a case of Hanot's cirrhosis which has become arrested.

Secondly, there is another class of cirrhosis which is known as *hepatic infantilism*. This is a very remarkable condition, and very rare. The liver is enlarged and cirrhotic, and the spleen is enormously enlarged, quite out of proportion to the liver, indeed, it may fill the whole abdomen. Jaundice is present and is an early symptom. There is no ascites or any other symptom of portal obstruction. Sometimes the superficial veins of the abdomen may be enlarged, otherwise the patients are perfectly well, quite lively, and have a good appetite. Occasionally there may be a little pain or diarrhœa, and epistaxis sometimes occurs. A remarkable point about these patients is that they are stunted in growth. They are undersized and have very little muscular development, they are sexually immature, and sometimes clubbing of the fingers occurs and enlargement of the ends of the tibiæ and femora. A case of hepatic infantilism died at University College Hospital some time ago, and this patient died from coma. The post-mortem examination showed the liver to be cirrhotic and the type was the unilobular type.

Thirdly, one has to consider cirrhosis as a *sequel of acute specific fevers*. The evidence that acute specifics give rise to cirrhosis is not very weighty. In the first place acute interstitial hepatitis has been described, and undoubtedly it does occur in scarlet fever and in measles, and it has also been described as occurring in association with small-pox. It is an infiltration of small cells around the bile-ducts and blood-vessels. In the second place, occasionally symptoms of cirrhosis may date from the fever itself. For instance, jaundice has commenced during an attack of measles and scarlet fever, and has been followed by a definite cirrhosis. In the particular case which I am referring to, death occurred from coma, and the post-mortem examination showed a cirrhotic liver one year after an attack of measles. That is fairly strong evidence.

Thirdly, a history of acute specifics may be obtained, as in this case, in a patient with cirrhosis. But, as a matter of fact, I think this is very poor evidence. You can get a history of measles or scarlet fever in a large number of children, and in this case I do not think the measles when he was a boy had anything to do with the present condition. So on the whole the evidence is not very great. Another point is that alcohol, which is commonly administered during acute specifics, sometimes largely by certain practitioners, may be responsible for the cirrhosis in some cases. The clinical type of disease in the cases which follow acute specifics on the whole appears to be of the hypertrophic, the Hanot type, jaundice being a very marked symptom. Ascites has been described in some cases as well.

There is a fever in which cirrhosis undoubtedly does occur, and that is malaria. These malarial cases are stated to always come from Africa. In England one practically never, so far as I am aware—and the same remark applies to America—sees a case of cirrhosis of the liver due to malaria. All the recorded cases appear to come from Africa. There is an enlargement of the liver and spleen, the patient is suffering from malarial cachexia, and the cirrhosis is of the multilobular type.

The *fourth class* of Type I includes *infantile biliary cirrhosis*. This disease occurs in lower Bengal. It is an Indian disease; sometimes it is called Indian cirrhosis. There is a superstition among Hindoos that this liver really is a turtle. They think the organ is endowed with life and that it must be killed by fire, and they accordingly come to the doctor to ask for a post-mortem examination so that the liver may be burned. This disease affects natives, very rarely Europeans, and it appears to run in certain families. There is no known cause for it, no history of syphilis or of malaria being obtained. It has been suggested that the condition may be brought on by improper feeding. I believe it is the custom of the Hindoos

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to give their children a great many condiments and spices, and it may be that these are at all events a factor in the causation. The disease begins at about the seventh or eighth month of life, and it is never seen after the third or fourth year. Its duration varies from three to twelve months. Death occurs from one of two things: either coma or hæmatemesis. The prognosis is not very good. It is stated that only about 6 cases out of 400 recover. With regard to the symptoms, the first is enlargement of the liver, and there are no other symptoms until the liver becomes enlarged. Then the child begins to suffer from nausea and vomiting, and slight jaundice with constipation. The motions are pale and the urine contains bile. These symptoms steadily develop with fever. The liver on examination is found to be enlarged down to the umbilicus, and the spleen also enlarged. The superficial veins in the abdomen are commonly enlarged. Later on intense jaundice occurs; it is slight at the beginning, and later becomes intense, and ascites may occur. In a few cases there is hæmatemesis and melæna, and occasionally subcutaneous œdema. At the post-mortem examination an interlobular cirrhosis is found, the liver-cells are destroyed, and there is a formation of new bile-ducts. As a rule the liver is larger than normal.

The *fifth condition* is cirrhosis associated with *congenital obstruction of the bile-ducts*. In these cases jaundice may not be present at birth, it may not appear until a few days after birth, although the bile-ducts are completely obstructed. The duration of life may be anything up to nine months. In all these cases the liver is found to be cirrhotic, and death is due to cholæmia or hæmorrhage, or to gradual wasting and exhaustion. Now, it is a well-known fact that cirrhosis may be present at birth without there being any obstruction of the bile ducts, and it is commonly thought that these cases are usually of syphilitic origin. The probability is that in this class of case the cirrhosis is the primary condition and the obstruction to the bile-ducts is secondary, because marked cirrhosis of the liver is probably never dependent upon obstruction of the bile-ducts. Clinically, obstruction of the bile-ducts does not lead to cirrhosis. There may be slight cirrhosis if the bile-ducts are also infected. What usually happens is that the bile-ducts become distended with clear mucus, the liver-cells become atrophied, and unless there is infection of the bile-ducts, such as occurs in chronic obstruction by gall-stones, there is no evidence of cirrhosis. Experimentally, the results do not agree. One can produce cirrhosis by ligature of the bile-ducts in an animal, but it is a very difficult thing to exclude infection of those ducts. Therefore experimental results are somewhat discordant. It would certainly be a very curious thing if congenital obliteration of the bile-ducts should give rise to cirrhosis whilst other forms of obstruction do not. It is a very significant thing that the obstruction is never found without cirrhosis. Again, the great enlargement of the liver and spleen much more simulate biliary cirrhosis than they do enlargement of the liver as the result of biliary obstruction. So altogether it is very probable that the cirrhosis of the liver is the primary condition and that the obstruction of the bile-ducts is secondary.

Sixthly, we come to alcoholic cirrhosis. It is very uncommon in children. So far, only seventy-four cases have been described in the literature. It usually occurs between six and nine years of age. The youngest case on record, I think, is about sixteen months of age. The disease is practically not seen in infancy. Males are more affected with it than females.

Now a word with regard to how the child gets the excess of alcohol. It is a common practice of parents to give their children alcohol for minor complaints, whatever the disease may be. One commonly sees parents giving children beer to drink: they say, "what is good for me cannot be bad for my child." Drunkenness may be acquired by example, the child having drunken parents. Many practitioners, as you know, prescribe alcohol in acute diseases, and parents sometimes go on giving the alcohol when the child is better. Lastly, dipsomania has been seen in children. There is a case on record of a child twelve years of age having been brought into a hospital dead drunk. So there are many ways in which children can become alcoholic. Again, children are much more susceptible to alcohol than are adults, and the same applies to animals. It has been shown that a single dose of alcohol may produce changes in the liver. Cirrhosis has actually been seen in a child after six months' administration of alcohol. I do not know whether you are aware of it, but alcohol affects the nervous system of children exactly the same as it

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does that of adults. There may be peripheral neuritis, alcoholic insanity, or delirium tremens in children. So it is not at all surprising that alcoholic cirrhosis should occur in children. The post-mortem appearances of alcoholic cirrhosis in children—and I have a specimen of it under the microscope for you to see afterwards—are exactly the same as in adults. Pulmonary tuberculosis is very rare in children, whereas in adults you will remember it is by no means uncommon. Tuberculous peritonitis may be present in children, and another thing which is very remarkable is that a terminal suppurative peritonitis is by no means uncommon.

The symptoms in children differ somewhat from those in adults. Jaundice is much commoner in children than it is in adults. On the other hand, hæmatemesis is less frequent in children than in adults, but hæmatemesis may occur in children, and it may be the direct cause of death. Epistaxis and skin hæmorrhages are quite as frequent in children as they are in adults. Diarrhœa is much more often seen, and you may also see in children repeated vomiting. The liver and spleen are usually very large, and ascites is very constant. There may be enormous ascites. Elevation of temperature is perhaps commoner in children than it is in adults. The presence of distended venules of the face and a sallow complexion are very important, and quite characteristic in children as they are in adults.

Certain peculiar nervous symptoms have been described in alcoholic cirrhosis in children. They are very rare, and they differ from the adult form in this respect: In the latter peripheral neuritis is fairly common, but in children this never occurs. But one does occasionally find certain nervous symptoms present. They consist of a spastic rigid condition of the limbs, which leads to contracture. There is no actual paralysis of the muscles. Mental weakness or idiocy may be present. Loss of speech is common, but the patient understands what you say to him. The reflexes and the electrical reactions are perfectly normal. At the post-mortem examinations which have been made on these cases, small softenings in the lenticular nucleus have been found. The relation which they have to cirrhosis of the liver seems very problematical. Otherwise nervous complications are practically unknown in children. A very important point in connection with alcoholic cirrhosis in children is that in the beginning the symptoms are particularly latent. The child suffers from no symptoms at all for a long time, and then after definite symptoms have occurred he dies very rapidly. It seems as if the child resisted up to a certain point and then gave way suddenly. Such cases of alcoholic cirrhosis as I have described are commonly diagnosed as tuberculous peritonitis. Tuberculous peritonitis is the commonest cause of ascites in children; in fact, if one gets a case of ascites with fever and wasting and a history of tuberculosis one commonly diagnoses tuberculous peritonitis. Again, an enlarged liver may be mistaken for an omental tumour occurring in tuberculous peritonitis or *vice versâ*. The dilated venules on the face and the slight jaundice which may occur are evidences in favour of cirrhosis. If you can feel a hard liver, the case is probably one of cirrhosis; if you can feel masses in other parts of the abdomen, it is probably a case of tuberculous peritonitis. These cases also closely resemble cardiac disease in children, especially where there is adherent pericardium, of which there may be no signs whatever. You know, of course, that the dropsy of heart disease in children commonly begins in the peritoneum, and the liver of children with heart disease becomes very much larger relatively than in adults. Wasting is a very prominent symptom in heart disease, especially mitral stenosis. So the similarity may be very great. On the other hand, in cirrhosis of the liver, œdema of the legs may occur, and you may find albumen in the urine. The points I would lay stress on in the diagnosis are—distended venules of the face, jaundice, the spleen being enlarged, larger relatively than in heart disease, and the systematic examination of the urine. There is a third condition which may be confused with alcoholic cirrhosis, and that is albuminoid disease of the liver, and of this last condition I have lately seen three cases, all of which had enormous ascites; they were all cases of spinal caries. The albuminoid disease was directly due to that. Of course, these patients have marked wasting as well. Ascites is not particularly common in albuminoid disease in adults, but in children it seems to be much more frequent.

Seventhly, there remains to be considered *congenital syphilis*. Pathologically syphilis may affect the liver in several ways. The commonest of all is by an inter-cellular infiltration with small round

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cells separating the individual liver cells from one another. Secondly, the condition may be due to scattered gummata and to the scars which are left by gummata. Thirdly, it may be a syphilitic peri-pylephlebitis. This gives rise to thickening of the connective tissue in the portal canals. Fourthly, all signs of syphilis may have disappeared altogether, and the patient may develop a condition which is exactly like atrophic cirrhosis. This has been called para-syphilitic cirrhosis. The probability is that the liver is altered in some way, so that cirrhosis may easily supervene. The inter-cellular variety is the usual type in infants. You may find, however, gummata with fibrosis even in newborn children. All the varieties I have told you of may be seen in the premature fœtus or in a full-term child, or during the first few weeks of its life. There is another condition, known as late hereditary syphilis, in patients of about eight or nine years of age, and you may find cirrhosis of the liver in this. The change is gummatous, that is to say, gummata with scarring. The symptoms of this condition are as follows: There is wasting and anæmia and other signs of congenital syphilis, the liver is enlarged and hard, and usually the spleen is enlarged also. In infants, jaundice may be present at birth, or it may appear within the first few weeks of life. Occasionally the subcutaneous veins are prominent, as in cases of portal obstruction, but ascites is very rare. In older children ascites is much commoner, and jaundice is rarer. Sometimes infantilism may be seen, with clubbing of the fingers, and occasionally albuminoid disease of the liver is present as well. The diagnosis, as a rule, is quite easy, because one has the other signs of syphilis present, and there is a history of that disease.

With regard to prognosis, the disease is very amenable to mercurial treatment. I do not think that the case under consideration is a case of syphilis, because there are no signs or symptoms of that disease, and there has never been any history of them. The clinical course has not corresponded to that of syphilis either, and pathologically the sections are seen to be different from those of a case of congenital syphilis.

Turning now to Type II, in which ascites is the main symptom, you will see that it includes the capsular cirrhoses in which there is very little cirrhosis of the liver tissue proper. The first condition I will mention is *adherent pericardium with chronic mediastinitis*. The pericardium may be simply adherent, or it may be calcified, or it may be tuberculous. Two conditions of liver are associated with this: first, chronic peri-hepatitis, and secondly, the condition known as pericarditic pseudo-cirrhosis. *Chronic peri-hepatitis* is sometimes known as sugar-iced liver. The general peritoneum is usually affected as well as the peritoneum over the liver. If a multiple affection of the serous cavities occurs, that is to say, chronic peritonitis, adherent pericardium and chronic pleurisy, the condition is sometimes called Concato's disease. Another name for it is multiple serositis or chronic polyrrhomenitis. I have sections of the liver from such a case under the microscope. The liver is covered by an enormously thick white capsule, with small pits on the surface; it is tough and strips off easily from the liver, leaving a smooth surface underneath. The liver itself may be compressed and deformed, but the substance of it is fatty, atrophied, and congested. The cirrhosis, however, is only of slight degree. The general peritoneum is thickened, the mesentery is contracted up, and the intestines dragged to the back of the abdomen and shortened. The stomach is diminished in size, and the omentum is atrophied. Sometimes the changes are not so extensive, and may be more or less limited to the liver.

With regard to the origin of the affection, it has been supposed to be a direct spread from the chest, mediastinum and pericardium. This condition may be readily recognised if there are present the signs of adherent pericardium, but only too commonly in this affection such signs are absent. The little girl, a section of whose liver I have under the microscope, had no signs of adherent pericardium. The apex of the heart was in the normal place, and there were no murmurs nor any indications of cardiac enlargement. The only sign present—and it is present in all these cases—was recurring ascites. Some patients are tapped over and over again for years. *Pericarditic pseudo-cirrhosis* is different from the last-named condition. The pericardium is adherent, but the cirrhosis of the liver occurs on the top of prolonged venous congestion. It is not due to spread through the diaphragm, and the cirrhosis is commonly only marked in the superficial layers of the liver.

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It is a cirrhosis on the top of a long-standing venous congestion. The capsule may be thickened, and there may be adhesions between the capsule and the diaphragm. Sometimes there is peritoneal thickening. The symptoms are similar to those of chronic peri-hepatitis, the liver is felt to be hard, and recurring ascites is the chief sign.

The distinguishing points between these two conditions and alcoholic cirrhosis are as follows: There may be signs of adherent pericardium. The spleen is more enlarged proportionately in portal cirrhosis than it is in the other conditions. In portal cirrhosis the ascites does not recur to the extent that it does in the other cases. The occurrence of ascites in cirrhosis means that the end is near, but in the other cases it does not mean anything of the kind; the patient may be tapped over and over again for years before death occurs. The character of the fluid obtained from the abdomen is different in the two conditions. In the capsular variety it is of a higher specific gravity, it is more milky in colour from intact or broken-up cells, and there is more albumen in it. It is an inflammatory exudate. Jaundice and other liver symptoms may be present in portal cirrhosis, but they are not present in the capsular variety, in which the patient does not die from toxic symptoms. Lastly, there is a history or evidence of alcohol in the child, which is of very great value in diagnosis.

There is not much to be said about cases of *cardio-tuberculous cirrhosis*. It is merely a secondary infiltration with tubercle of a liver which has been congested for a long time. The infiltration occurs along the portal spaces, and the tuberculosis itself is not the cause of the cirrhosis. The cirrhosis is present, and this tuberculous infiltration occurs on the top of it.

The third variety of this class is *cardiac cirrhosis*. The cirrhosis here is secondary to chronic venous congestion, the result of heart disease. There is no doubt that a certain amount of fibrosis is formed in the liver as the result of heart disease, but it never occurs to the same extent as in atrophic cirrhosis. It is only very slight. The liver is enlarged and somewhat tender, and the spleen may be enlarged to some extent, though not so much as in portal cirrhosis. Ascites is present and there is wasting to a marked degree; jaundice is absent, though there may be a slight tinging of the conjunctivæ. A fourth variety is associated with *obliteration of the hepatic veins*. This is very rare. Only very few cases have been described. The hepatic veins anastomose with the diaphragmatic veins, and the portal vein with the veins of the umbilical ligament. The liver is cirrhotic, and the probability is that the cirrhosis is the primary condition, and that thrombosis of the hepatic veins has occurred and has led to fibrous occlusion. With regard to symptoms, the child has pain and the abdomen is swollen; ascites comes on and, after tapping, re-accumulates. The spleen has been found normal and there is no jaundice or bleeding. Of course, this condition cannot be diagnosed; it is really a pathological curiosity.

Type III is that in which the liver is found to be enlarged in a patient who is suffering from some disease likely to cause it. The first division of this class includes *rickets*. The hepatic enlargement is merely a temporary condition and is of no importance. The liver shows a certain amount of fatty change, and there may be some fibrous change also in the portal spaces. The spleen is often felt below the costal margin and the child is found to be rickety. Secondly, there is *syphilis*. Although, as I have mentioned, syphilis of the liver usually gives rise to some hepatic symptoms, the only symptom or sign may be the presence of enlargement of the liver, that is to say, a child suffering from congenital syphilis may have merely an enlarged liver without any sign of portal obstruction or of hepatic insufficiency.

Thirdly, tuberculosis of the liver may be associated with cirrhosis. In miliary tuberculosis and also in the chronic forms of that disease there is certainly some formation of fibrous tissue. But such a condition does not give rise to true cirrhosis, or if it does, it must be very rare indeed. Cirrhosis of the liver may be dependent on the tuberculous process, and it may extend from tuberculous peritonitis. But even though it extends from tuberculous peritonitis the cirrhosis is a simple cirrhosis. No tubercles are found in the liver in this condition, portal cirrhosis is present, and the fibrous tissue does not extend between the cells; the capsule may be thickened, there may be some adhesions, and the coats of the arteries and veins may be thickened. In addition to this you find tuberculous peritonitis with matting of the omentum and intestines. Caseous mesenteric glands with tuberculous ulcera-

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tion of the intestines may be present and the child may have tubercle of the lungs or tuberculous meningitis. Clinically, the liver may be felt, or it may not be. It gives rise to no direct liver symptoms. So one can make the statement that tuberculosis, whether it occurs in the form of tuberculous pericarditis and mediastinitis or tuberculous peritonitis, gives rise to a simple cirrhosis in the vast majority of cases, the cardio-tubercular cirrhosis being merely a secondary infiltration with tubercle on the top of a cirrhotic liver.

Fourthly, I will merely mention *splenic anæmia*. In that condition the liver is very commonly enlarged, and it is found to be slightly cirrhotic. It is supposed that possibly some poison may be manufactured in the spleen and passing to the liver give rise to cirrhosis. Commonly there are no signs or symptoms, at all events in the early stage, except those due to anæmia. The liver is enlarged, and later on ascites may develop.

December 2nd, 1907.

Hyoscine Hypodermic Anæsthesia.—F. B. Kirby follows the plan herewith given. The patient is given a hypodermic injection of hyoscine hydrobromide $\frac{1}{100}$ gr., morphine hydrochloride $\frac{1}{4}$ gr., and cactin $\frac{1}{67}$ gr. The room is darkened and the patient encouraged to sleep. One hour later a second dose is given and in half an hour the operation may be begun. If the patient is not completely "under," a little chloroform will suffice. The writer's experience has been mainly in obstetrics, and he notes that the patient usually sleeps between the pains, which always awaken her to active help in aiding expulsion. In some few cases the child has been cyanotic for a while, but always revived in a short time. This effect on the child is avoided by omitting morphine from the second dose. In no case have any untoward results been observed. The anæsthetic is too profound for minor surgery and should be used with care at the extremes of life. Dr. Kirby declares that in railroad and other accidents the new method is of great value. Here twenty-five or fifty people could receive preliminary pain-relieving treatment while one was being cared for with ether anæsthesia and in the hour and a half necessary could be transported to a suitable place for care.—*Medical Record*, vol. lxxii, No. 20.

THE POSITION, DEFORMITIES, AND INJURIES ASSOCIATED WITH ABDUCTION OF THE FOOT.*

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Lecture II.

In the previous lecture we were concerned with the adducted foot; in this one we shall deal with the abducted foot. It is the position of rest or inactivity as contrasted with the adducted position of activity. Both in fact and in terminology each is the exact converse of the other. Like adduction, abduction of the foot is not a simple movement; almost all the joints of the tarsus participate in it, particularly the mid-tarsal. It is the position which the foot assumes when in rest or inaction, and should its assumption become a habit, as it is particularly liable to in young and weakly subjects, the position becomes more or less fixed and permanent, to the very great athletic disadvantage of its possessor. It is associated with the deformities of talipes calcaneus, talipes planus, talipes valgus, hallux valgus, hallux rigidus, hallux flexus, digiti flexi and rigidi. This series of positions or deformities is precisely the opposite to that associated with the adducted position, see previous lecture. Hence the deformities of the foot can be arranged in two distinct series, each being the converse of the other, the various conditions being no longer isolated from each other.

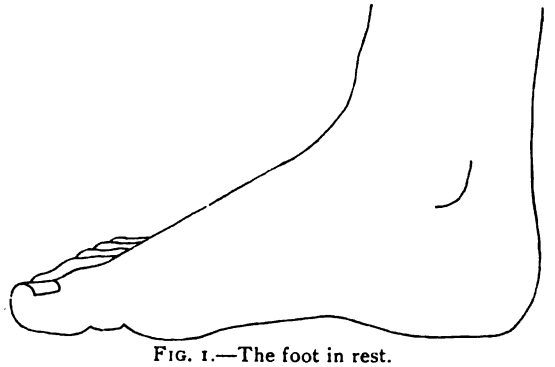

Fig. 1.—The foot in rest.

* Post-graduate lecture, delivered at the Polyclinic and illustrated by lantern slides.

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Before proceeding to the immediate subject, it is desirable that some points should be indicated by means of which the peculiarities of the abducted foot are readily recognised.

(1) Inactive, young or weakly people turn their toes out. As people grow old their feet naturally assume the abducted position which gives the peculiarity to their gait, such as is caricatured by the shuffling movements of the aged.

(2) Those who habitually turn their toes well out have abducted feet.

This demonstrates the conjunction of eversion of the foot with flattening of the arch.

(3) Such people often do not lift their feet well from the ground. The abducted position lends itself to a shuffling gait.

(*a*) Talipes calcaneus.
(*b*) Eversion of the foot, talipes valgus.
(*c*) A flat arch, talipes planus.
(*d*) Hallux flexus and its rigidus.
(*e*) Hallux valgus.
(*f*) Digiti rigidi and flexi.

Only a few of these conditions merit special mention, and such as do will be considered *seriatim.*

Talipes.—The varietes of calcaneus, valgus and planus are so invariably combined in the acquired deformities that it would be pedantic to attempt to consider them separately. When in the congenital form it is otherwise.

The resting position of the foot is acquired very largely from the weakness or paralysis of one

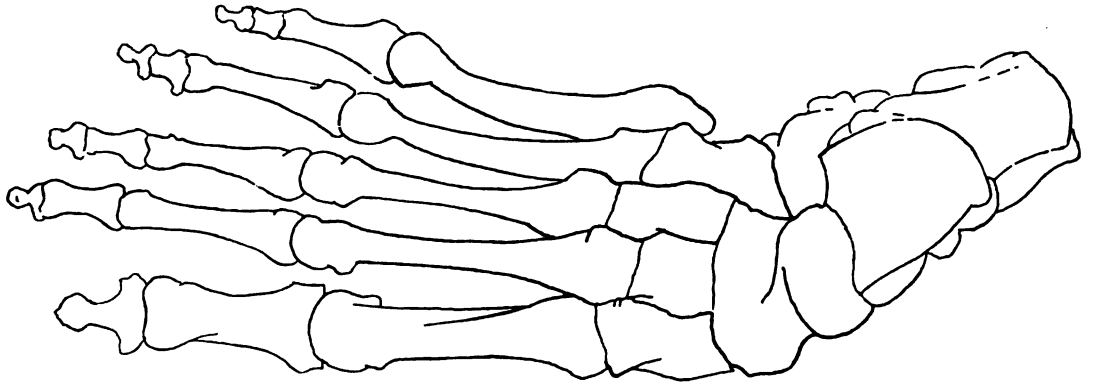

FIG. 2.—The bones of the abducted foot.

(4) The upper of the boot is long and has no creases across the "toe."

(5) The foot is a long one, frequently thin and narrow.

(6) The heel of the boot is worn on the outer side, because the foot is brought forward abducted and imperfectly raised clear of the ground.

(7) The callosities on the sole are on the outer side of the heel, and under the ball of the third toe, sometimes under the fourth and fifth. The skin under the ball of the great toe is thin and silky, whilst distal to this under the head of the proximal phalanx a corn can often be found.

(8) These people stand firmly and steadily on heir legs, provided that their general bodily condition allows of it and quick, alert movements are not required. It is the position of the slow, heavy and inert.

(9) The deformities associated with it are:

muscle, reference to which has been made in the preceding lecture. It has been demonstrated that contraction of the tibialis posticus raises the instep and the arch of the foot by approximating its insertions upon the tubercle of the scaphoid and the sustentaculum tali of the calcaneum. When weak or paralysed these two bony points will become separated from each other by the action of the body weight. The other muscles in the calf and the sole of the foot cannot replace a feeble or paralysed tibialis posticus; all they do and are capable of doing is to coordinate and balance the movements at the tarsal joints, so as to enable the tibialis posticus to act with mechanical advantage on a many-jointed lever. Just as when speaking of pes cavus stress was laid on the weakness or paralysis of the peronei muscles, so in speaking of the opposite condition of pes planus stress is laid on their great antagonist, the tibialis pos-

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ticus. There is a fact that must be borne in mind in treating these cases. The usual method adopted is by means of physical exercises, which unfortunately may strengthen the peronei at the same time as they are strengthening their antagonist, the tibialis posticus; the peronei preventing the latter muscle from raising the arch. The method of physical exercises has never been taught with reference to the physiological positions of the foot. When the foot is abducted into the inactive or resting position, the peronei cease to be evertors or "turners out" of the foot, becoming flexors of the ankle-joint with the calf muscles. Hence exercises taught in the ordinary way will merely strengthen them, thus preventing inversion and adduction of the foot. The movement of inversion should be practised both actively and

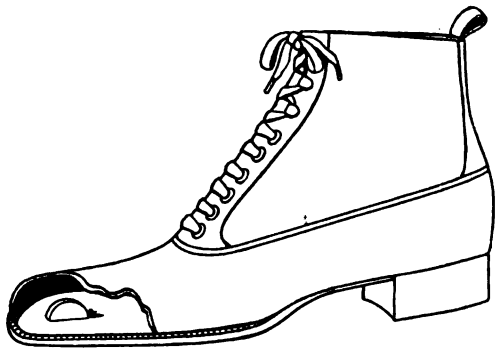

FIG. 3.—Showing long straight upper, increased. Incidentally it shows a "toe post."

passively. And the tip-toe exercises must be taught with the feet directed straight forward or even with the toes turned in. The question as to whether the peronei tendons require division or lengthening must be decided early. Should the tendons resist passive inversion of the foot by means of manipulations by the surgeon, particularly if the patient is under an anæsthetic, then the tendons had better be divided. In doubtful cases let the exercises be pursued for a month under a competent instructor, at the end of which time the case must be examined again and the rate of progress considered. The question can then be decided.

When speaking of pes cavus, it was advised that some paralytic cases offered one of the best opportunities of successful tendon grafting. With regard to pes planus or the paralytic abducted foot this is not so. In certain cases of anterior poliomyelitis, where the paralysis is very partial, it is possible to use a piece of the tendo Achillis and thread it on to that of the paralysed tibialis posticus. But it is unusual. The other tendon graftings are not nearly so successful, such as putting part of the peronei or tibialis anticus on the tibialis posticus.

Hallux valgus.—This condition is generally found in association with the abducted position of the foot. It is seen most frequently in ladies' feet, which, owing to their peculiarities, merit special consideration by themselves, Lecture III. In them it is often combined with some extension (dorsal flexion) of the toe at the metatarso-phalangeal joint. When present with an abducted foot, the toe is usually stiff, with power of flexion but little or none of extension—the hallux rigidus of the abducted foot. The deformity is part and parcel of the position of abduction. In fact, it may begin in that way, the big toe being abducted with the rest of the foot, and, as the arch flattens, the toes are pushed further down into the boot, which, if unsuitably made, exaggerates the valgus position. On account of the pressure exerted by the head of the metatarsal against the "upper" of the boot, a bony malformation is developed on the upper and inner part of the head of the metatarsal. As this boss of bone grows it increases the hallux valgus and rigidus. Thus it is easy to derive the hallux valgus deformity from the abducted position. Its presence in the female foot will be considered in the third lecture.

Owing to the friction to which the bony boss is subjected a bursa is developed over it, the inflammation of which is responsible for much of the pain connected with bunions.

The presence of a hallux valgus with an inactive or abducted foot increases the difficulty which the possessor has in getting alertness and activity out of his foot. No one with a hallux valgus can be alert and active, nor can run quickly and neatly.

Hallux valgus in the male foot is merely a symptom of its abducted position, so that permanent relief must be sought by treating the position and not the toe. Often it is necessary to do something to relieve the toe, particularly in women. A flap of skin must be turned down from over the bunion and the bursa removed; the bony boss itself is then removed with a chisel and mallet. But before concluding the operation a thin-bladed

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knife must be passed through the metatarso-phalangeal joint and the external lateral ligament divided so that the direction of the toe can be corrected. But it must be remembered that hallux valgus is only a symptom and not the whole disease. In consequence, after the operation the hallux valgus will return in a painless form.

A light splint should be worn for a few days or longer, the boots and shoes to be subsequently worn corrected, and if necessary a toe-post inserted. Care should not be remitted until an attempt has been made to correct or relieve the inactive abducted position of the foot by means of exercises. The treatment differs very much with the sex of the patient, so long as women wear high-heeled boots it will be impossible to convert their feet to the adducted position.*

Hallux rigidus.—Hallux rigidus is a name applied in general to a stiff and painful position of the big toe which is associated with the flat or abducted position of the foot. It is a very great handicap to its possessor, because in walking or running the big toe of the foot which is on the ground is extended, or dorsally flexed at the ankle-joint in taking a step forward. If the patient suffers from hallux rigidus his toe cannot be extended and therefore he is unable to take active exercise, to use tiptoe exercise, or to take any but short steps. Further, it is produced, as is indicated in Figs. 4 and 5, by the development of a boss of bone on the dorsal surface of the head of the metatarsal of the great toe, as the result of its pressure against the upper of the boot. The great increase of the size of the dorsal part of the head of the bone is easily observed clinically. Unfortunately for the patient the development of a hallux rigidus does not diminish the pressure of the "upper" of the boot, but increases it, so that the condition of hallux rigidus is not a final but a temporary one, which, as the head of the metatarsal bone becomes more and more misshapen, the toe is flexed more and more towards the plantar surface, becoming a hallux flexus.

In the previous lecture I have shown that there is a corresponding hallux rigidus associated with the adducted position of the foot, which was capable of extension (dorsal flexion) and not flexion (plantar), and which proceeded to hallux extensus, Figs. 8 and 9, Lecture I. The hallux rigidus of the abducted position is the precise opposite; the toe is capable of plantar flexion at the metatarso-phalangeal joint and not of extension (dorsal flexion). Further, the deformity will progress to hallux flexus, instead of to hallux extensus as in the previous instance.

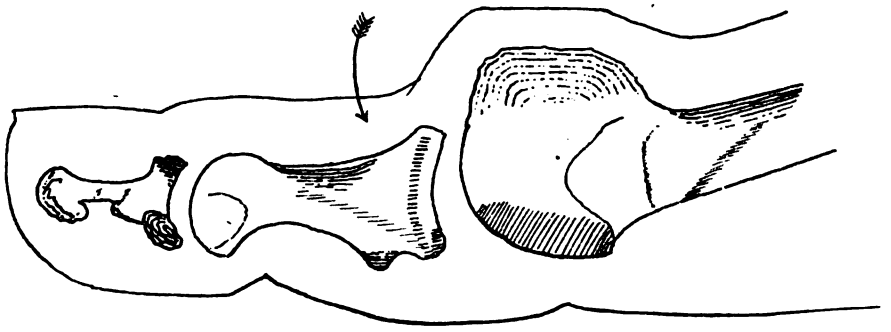

FIG. 4.—Hallux rigidus of the abducted foot. Dorsal enlargement of head of metatarsal.

Having described the characters of the hallux rigidus of the abducted foot sufficiently, it is advisable to consider its treatment. If there is any movement of extension (dorsal flexion) of the toe, it must be conserved and increased by means of massage and exercises. Movement under an anæsthetic sometimes does good, but too often is disappointing. "Tip toe" exercises must be taught, commencing with the foot in the "toes out" or abducted position which, as improvement takes place, is changed by degrees, bringing the toes more and more inward until the "toes in" or adducted position is attained. The greatest stress must be laid on the importance of persevering with this treatment, because an hallux rigidus of the abducted position of the foot will prevent its possessor ever being active and will make the abducted position of the foot worse.

* 'Operations of General Practice,' Oxford Medical Press, pp. 245–246 and 249–250.

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Thus its presence is the indication of a serious menace to the future activity of the person.

Some of these cases of hallux rigidus come to operation. The obvious operation is to remove the boss on the dorsal surface of the head of the metatarsal bone which is causing all the trouble. But this is unfortunately followed by indifferent success, due in part to the adaptive shortening of the soft parts. I have done it several times, and have abandoned it as it did not come up to my expectations. The next most obvious operation is to amputate the toe. But this is not advisable. In fact, if any operation has to be performed it should consist of excision of the head of the first metatarsal bone. This seems a severe measure when it is remembered how the anatomical text-books make such a tremendous point of its functional value in walking and standing. And so it is in the active and adducted foot. But a little reflection will show it to be otherwise in the abducted foot. The points of pressure will be indicated, as when a person stands, by the callosities present on the soles of the feet. To remind you of these I must refer you to figures which indicate them.* You will observe that in an adducted foot there is a callosity under the ball of the great toe and in the abducted foot it lies under the third toe, whilst the skin under the ball of the big toe is "soft and silky," and, therefore, sustains no pressure. Under these circumstances it cannot be any great loss if the head of the first metatarsal is removed in abducted feet. Though this is true from the point of view of bearing pressure, it is a procedure which should not be too lightly undertaken; it makes the "tread" of the foot narrower and the person in consequence less steady, and the foot permanently flat or abducted. Hence, this operative measure must only be undertaken when the patient's pain and disability are so great as to prevent them taking exercise or following their occupation.

It absolutely precludes that patient's foot ever assuming the active adducted position. But then if a person, over twenty say, has a foot which has become fixed or set in the abducted position, there is no means by which that fixed abducted attitude can be converted into one of activity and adduction. Hence, as the damage to the individual's usefulness is already done, it only remains for us to make the best "of a bad job." The best of a bad job is sometimes to remove the head of the first metatarsal bone.

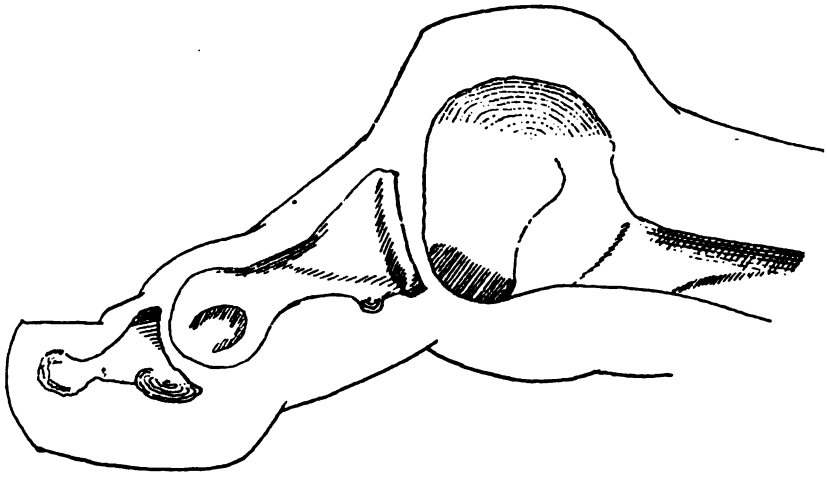

Fig. 5.—Hallux flexus. Exaggeration of the condition in Fig. 4.

When the foot is in the active adducted position there is a callosity under the ball of the big toe, and under such circumstances it will be a serious loss to the individual if the head of the first metatarsal bone is removed. So that it may be said that this operation is one whose performance is associated with the abducted position; and that it should rarely, if ever, be performed on an adducted and active foot.

Hallux rigidus and its hallux flexus often are not

* Clinical Journal, August 29th, 1906, p. 314, Figs. 5 and 6.

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pure deformities, but are commonly complicated by some degree of two other deformities. One we have already discussed, hallux valgus, in which case the bony deformation of the head of the metatarsal, instead of being only on the dorsum is on the dorsal and inner or tibial side, thus producing at the same time both the hallux rigidus or flexus of the abducted position and some degree of hallux valgus. It is a noteworthy thing that the abducted inactive position of the foot is associated with both talipes and hallux valgus, whilst the adducted active position of the foot is associated with both talipes and hallux varus. Both have a suitable hallux rigidus, from which starting point the active and inactive positions branch into the deformities of hallux extensus and hallux flexus respectively (Fig. 9 in previous lecture and Fig. 5 in this).

The association of hallux valgus with an arched

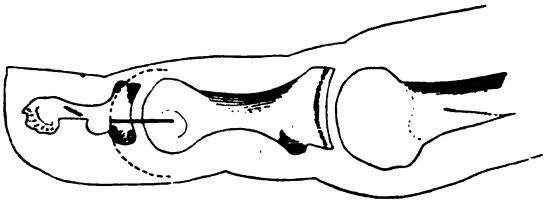

FIG. 6.—Diagram showing range of movement natural to the interphalangeal joint of the great toe.

foot is an unnatural and purely artificial condition which will be discussed when the female foot is considered.

Hallux flexus is a further stage of the hallux rigidus of the inactive abducted position of the foot, in which the big toe is bent into the sole of the foot by an increase of the bony boss on the dorsum of the head of the metatarsal, and is capable of some plantar flexion but of no dorsal flexion or extension. When the deformity is present in any degree of severity the disability caused by it is so great that palliative treatment is already quite out of the question. For practical purposes there is only one operation to be performed, because its results are so much better than any of the others suggested. This is amputation of the big toe at its metatarso-phalangeal joint.

Digitus extensus of the great toe.—The second deformity commonly associated with the hallux rigidus—hallux valgus was the other—of the abducted position is an extension or dorsal flexion of the interphalangeal joint. It is a deformity which has been classed amongst those due to arthritis of the joints by the late Mr. W. Anderson.* Osteo-arthritic changes are certainly found in the inter-

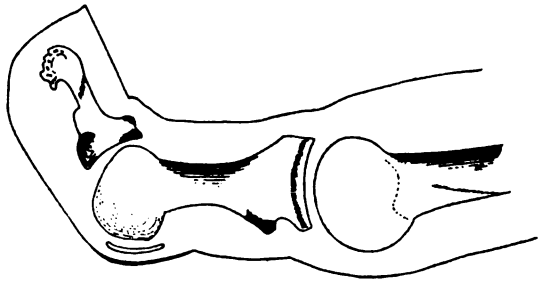

FIG. 7.—Digitus extensus of the great toe. The extension or dorsal flexion of the great toe has taken place in consequence of the development of the bony boss on the plantar surface of the head of the proximal phalanx. A corn and bursa under this boss are indicated.

phalangeal joint, with marked bony outgrowths on the plantar part of the head of the first phalanx, which by their growth displace the terminal phalanx backward, extending or dorsally flexing it. A callosity develops under the head of the proximal phalanx and a bursa beneath it. This "corn" is frequently a source of trouble in abducted feet and it may cripple the patient. The accompanying figures show the method of production.

In the majority of cases palliative treatment for the abducted foot and hallux rigidus must be tried and is all that is required. But sometimes the disability of the digitus extensus of the great toe is so

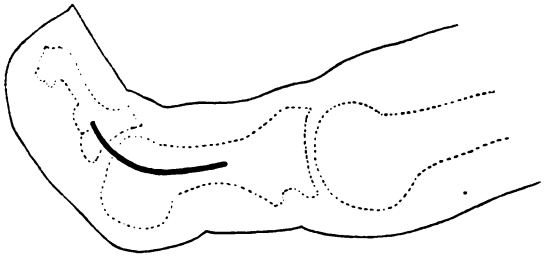

FIG. 8.—Diagram similar to Fig. 7, with lateral incision for the removal of the head of the proximal phalanx for digitus extensus of the great toe.

great and cripples the patient to such an extent that something must be done. In these cases the head of the proximal phalanx of the great toe is to be removed through a lateral incision, so as to avoid the corn. The pain and disability will have

* 'Fingers and Toes,' Churchill, p. 124, *et seq.*

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been relieved, but so long as the foot remains in the abducted position, which is usually for the remainder of the patient's life, the deformity will tend to, and probably will, recur. But if it does it

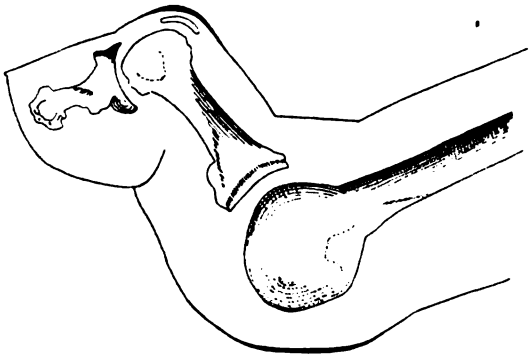

FIG. 9.—Digitus flexus of the great toe of the adducted foot, formerly called hallux retractus. A bursa and corn are shown on the dorsum.

will be unaccompanied by the pain and disability which previously resulted from it, owing to the removal of the head of the phalanx.

Connected with the hallux extensus of the abducted active position of the foot has been described a deformity, the reverse of the digitus extensus of the great toe just discussed, under the name of *hallux retractus*. But I would rather call it the *digitus flexus of the great toe*, because it is associated with the hallux rigidus and extensus of the active position of the foot, and is the antithesis of a corresponding condition in the inactive foot. It rarely requires special treatment, but a painful

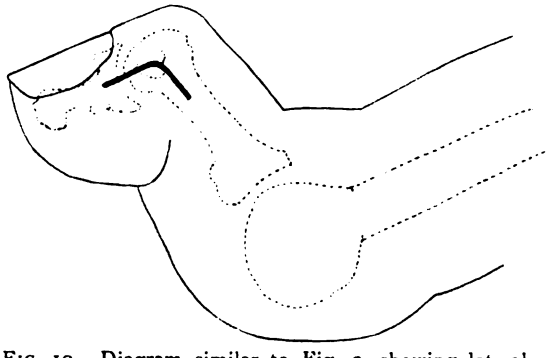

FIG. 10.—Diagram similar to Fig. 9, showing lateral incision for the removal of the head of the proximal phalanx.

corn may be developed on the dorsum of the head of the phalanx requiring operative relief, which is to be afforded by the removal of the head of the proximal phalanx through a lateral incision.

Digiti, rigidi and flexi.—With the inactive abducted position the toes take up a corresponding condition to that which the hallux assumes. They lose their power of extension or dorsal flexion, becoming digiti rigidi, and may get slowly bent towards the sole of the foot, digiti flexi. Occasionally one of the interphalangeal joints becomes dorsally flexed (extended) into a condition comparable to the digitus extensus of the great toe (Figs. 6, 7, and 8). The consideration of the condition of the little toe has been reserved for a sequel to these lectures on the consequences of wearing high heels.

There is a condition closely associated with these deformities of the toes which must be referred to

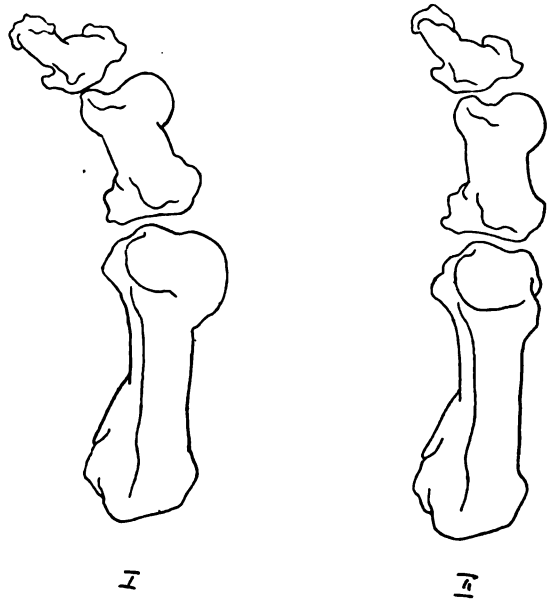

FIG. 11.—I. Digitus valgus of the great toe, the fibular deflection taking place at the interphalangeal joint and due to the formation of a boss on the inner or tibial side of the head of the proximal phalanx. It is combined with hallux valgus at the metatarso-phalangeal joint. II. Digitus valgus of the great toe, uncombined with any hallux valgus at the metatarso-phalangeal joint.

here. It is called after its appearance *ingrowing toe-nail*, and is found on the inner or tibial side of the great toe. As great pain and disability may be produced by it, it is important and necessary that we obtain a clear conception of its relations. First, it is commonly associated with the inactive abducted foot. Secondly, it is commonly associated with a great toe which is deficient in the power of extension, or dorsal flexion, at the metatarso-

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phalangeal joint — the hallux rigidus of the abducted foot. Thirdly, it is commonly associated with some degree of hallux valgus, which may arise either at the metatarso-phalangeal or the interphalangeal joint of the great toe, or at both. Fourthly and lastly, it is often associated with some degree of digitus extensus of the great toe, which is somewhat extended at the interphalangeal joint. The last two associations are responsible for the ingrowing toe-nail, namely the valgus and extensus at the interphalangeal joint, by pressing the great toe up against the upper of the boot, or far less frequently its neighbour the first toe, which is straighter and more rigid than usual—the digitus flexus of the abducted foot. Hence the skin and flesh on the inner or tibial side of the great toe is squeezed against the nail and sets up some septic ulceration, producing the painful condition of ingrowing toe-nail.

This explanation gives the key note to the palliative treatment, namely, treat the inactive abducted position of the foot of which the ingrowing toe-nail is a result. Incipient cases get quite well under this treatment. Later it may be advisable to remove the inner or tibial half of the nail with its matrix and the "in-growing" edge of the skin. In any case exercises should be taught after the operation. This little operation is described and illustrated in my book on 'The Operations of General Practice,' written in conjunction with Dr. Irving Pinches, pp. 246–247.* But as the deformity of the foot and toe is left untouched and may not be stationary but progressive, this little operation may only be successful for a time. Then, rarely, it will be necessary to proceed to further measures, such as removal of the head of the proximal phalanx.

Associated deformities.—In the previous lecture reference was made to some common deformities elsewhere in the body which were found associated with the active and adducted position of the foot. It was pointed out that with the adducted position the ankles tended to bend outward; with the inactive abducted position they bend inward, forming the very common condition in children called *weak ankles*. Attention was also drawn to the fact that in the former the larger external malleolus prevented the outward displacement, whilst in the latter the foot is everted and the ankle bulges in, the internal malleolus being insufficient to prevent it. Sometimes it is the result of a paralytic lesion affecting the tibialis posticus muscle alone or in a greater degree than the other muscles of the calf. In general, it is the result of the development of this muscle being insufficient for the requirements of the manner of life. *To treat the condition, this muscle must be educated, its action not replaced by special apparatus or boots.* It is a frequent thing to find these children and young adults already supplied with a heavy, strong boot. In these cases a heavy, strong boot is as bad for them as is a spinal jacket for an early case of lateral curvature, scoliosis. *The muscle must be educated, not replaced.* The best exercise for this is skipping on a mat without boots or shoes with the feet adducted; going upstairs on tip toe is another and very good one. It is appalling to think of the well paid and highly trained men who are employed to instruct the minds of young people, whilst the education of their bodies is entrusted to an old soldier, who has often left the service from inefficiency. So long as educated people are content to allow this stupid state of things to continue so long will our bodies suffer at the expense of our minds. It is like storing treasure in houses, some of whose walls are crumbling.

The treatment of cases of weak ankles requires great persistence and tact.

After weak ankles, *genu valgum* is the deformity most frequently associated with flat, abducted feet. It is a very common condition to find in its lesser degrees, each deformity being a cause for the other. At the hip-joint *coxa vara* is the inactive resting deformity.

The most important inactive deformities of the trunk often associated with abducted feet are the *round shoulders, winged scapulæ and kyphosis* which hold the chest in position of expiration, in which it will become fixed if nothing is done to prevent it. Now a boy's chest contains the most important elements of his athletic life, and the chest wall may be compared to the "bonnet" which encloses the engine which moves a motor-car. If the engine is bad the car cannot go well. Thus a person with a poor chest has a poor active or athletic capacity. This is the most important subject, and worthy of the consideration of the men who have the care of

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the young in their hands*. The men who had control of the young in former times had at last to be compelled to educate their minds, so history tells us. Let us hope that public opinion will soon compel them to attend more to the bodies. The old proverb, 'Mens sana in corpore sano,' should be ever before the eyes of the pedagogue so that they may yet take heed of it.

Injuries peculiar to the abducted foot.—In the previous lecture it was pointed out that there were injuries peculiar to the adducted foot which resulted from the application of violence to the sole where it has been accustomed to bear pressure, such as if a man fell straight on to the sole of his foot. The most frequent of these injuries was a fracture of the base of the fifth metatarsal bone. There are, similarly, injuries peculiar to the transmission of violence from the sole of the abducted foot. In this case the injury is most frequently found in the shafts of the second, third, and fourth metatarsals, and if there is no fracture of these there is often much pain in the neighbourhood of the external cuneiform bone. Whether the latter injury has taken the form of a fracture, partial dislocation, or injury to the soft parts (a sprain) it is often impossible to say, as the bones are so disposed that it is extremely difficult to obtain skiagraphs which show its nature.

Further, abducted feet are much more liable to sustain serious injuries, such as a fracture or dislocation, than are adducted feet.

December 2nd, 1907.

* Edred M. Corner, "The Importance of the Development of the Chest in the Young," 'Archives of the Roentgen Ray,' 1905, vol. lvi, pp. 219–223; also "The Physiology of the Chest in Exercise," *ibid.*, 1905, No. 63, pp. 133–135.

Iodide of Potassium.

The only danger in connection with the administration of iodide of potassium—and it is a serious one—is presented by the fact that its administration has been known to cause œdema of the glottis. Elsner and Huchard have both recorded cases of this kind, so that it is well to warn a patient to whom we are giving it for the first time to stop the medicine and report himself if any throat discomforts arise. Of inconveniences there are many. Perhaps the most pronounced is the occurrence of diarrhœa, either alone or accompanied by vomiting. This is not altogether infrequent, because the drug certainly has an irritating effect upon the gastro-intestinal tract, and should therefore be given with caution where this system is deranged. A form of iodism which has often given rise to unfortunate mistakes is that which causes pain and swelling in the parotid gland, accompanied by other symptoms strongly suggesting mumps. Ignorance of this fact may easily cause a deal of useless trouble and alarm in a household, and if the iodide is not stopped the condition will persist. The commonest forms in which intolerance shows itself are in the production of sore gums, sore throat, running at the eyes and nose, and metallic taste in the mouth. These vary much in degree, and may generally be checked by the addition of a few drops of Fowler's solution to each dose. Next in order of frequency come the skin manifestations of iodism. These are numerous; they may, indeed, take almost any form, but it is well to remember the purpuric form, especially when we are giving the drug to a rheumatic subject, for if we forget it it would be our duty to confine the patient to bed until some time after all signs of the rash had disappeared. True rheumatic purpura is just as liable to give rise to cardiac complications as acute rheumatism or chorea. The best way of giving iodide of potassium is in cachet form, followed by a draught of water. If given in solution over long periods of time it is well to add small doses of arsenic and nux vomica. The latter seems to prevent the occurrence of the mental and physical depression which some people experience when taking it for long. Sarsaparilla, liquorice, and syr. pruni virg. are said to disguise its taste. According to some writers whose opinions are eminently deserving of attention, the iodide of sodium is more easily tolerated than the potassium salt. Professor Huchard, who has great faith in the iodides, insists very much upon this superiority of the sodium salt, which he seems to think is to be preferred on more grounds than one. That iodism in all forms is much less liable to occur with large doses—*e. g.* 10 gr.—than with the ordinary dose of 2 gr. is a matter of common observation. The late Sir George Macleod was in the habit of teaching that if symptoms of intolerance occurred the right course was to double the dose, whatever its original amount.

The above is taken from an excellent work, published for Dr. Leonard Williams by Messrs. Baillière Tindall and Cox, entitled 'Minor Maladies and their Treatment.' The author's object in issuing this work has been to supply, in an accessible form, detailed information on subjects with which the ordinary text-book necessarily deals in a cursory manner. In the contents table is a list of subjects ranging from colds to insanity, and all these are treated by the author in an interesting and able manner.

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THE CLINICAL JOURNAL,

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A CLINICAL LECTURE

ON

BRACHIAL NEURITIS AND OCCUPATION NEUROSES.

Delivered at the West-End Hospital for Diseases of the Nervous System.

By THOMAS D. SAVILL, M.D.Lond.,
Physician to the Hospital.

GENTLEMEN,—Brachial neuritis is an affection in which the leading symptoms are pain and weakness in the arms associated with tenderness along the nerves and some of the other signs of inflammation of the nerves of the arms or forearms. In course of time some degree of atrophy usually supervenes. This man, æt. 58 years, who came to the hospital for the first time this afternoon, exhibits all the symptoms of brachial neuritis. He came to us for shooting and dull aching pains in the left arm, and there are numerous tender spots towards the end of the limb. The pain is increased by movement and he holds the arm quite still. You observe that there is marked tenderness when I press over the nerve trunks on the inner aspect of the arm, over the musculo-spiral at the back of the arm, over the median at the bend of the elbow and at the wrist, and over the ulnar behind the olecranon.

This tenderness is important. You may remember that the symptoms of peripheral neuritis were in former times regarded as "functional," and that many observers refused to believe in the existence of inflammation of the peripheral nerves until tenderness over the nerve trunks and their histological changes were demonstrated. The degree of tenderness and of pain depend, however, on the acuteness and severity of the neuritis; in some stages the pain is only slight and the tenderness ill-marked. You will notice that there is wasting of the forearm, a difference of nearly three quarters

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of an inch being observable. The nerves are too tender to permit of electrical examination. All his symptoms came on in June last, soon after a very severe carbuncle the scars of which you can still see across the back of the neck and the left shoulder. It is probable that the spinal nerves or nerve trunks of the left brachial plexus became involved in the inflammation. In this case one arm only is affected and the cause is obvious, but in the brachial neuritis of which I wish to speak both arms are usually affected, though one, usually the right, may present more pronounced symptoms than the other. Indeed, the patient may come complaining only of one arm, and it is only on inquiry we find symptoms in the other. Occasionally careful search may discover symptoms in the legs. Some of these cases come before us as writer's cramp, or some other kind of occupation neurosis. But it is of the highest importance to carefully examine such cases, because a bilateral affection points without much doubt to a toxæmia, combined in some cases with over-functioning or some other localising factor.

The private patient who has kindly consented to come here to-day, a married lady, æt. 55 years, consulted me about a month ago for weakness in both arms, so marked that the grasp recorded by the dynamometer was only a quarter of normal on the right side and one third on the left, and there were pain and tenderness along the courses of the nerves. Both forearms were thin, but the right, where all the symptoms were most marked, was three eighths of an inch less than the left. At that time there was no swelling of the wrist-joints such as there is now, but she complained of "creaking pains" in the shoulders, which made me suspect a rheumatic or gouty toxæmia. The illness had started very gradually about fifteen months ago with numbness in the finger tips, so that she could scarcely feel the needle when sewing. This was soon followed by pain in the fingers shooting up the arms and progressive weakness. Lately she has complained of some weakness of the legs. The knee-jerks are not lost but are slightly diminished, and suggest that the neuritis is spreading to the legs. Since she first came under observation a month ago the swelling of the wrist-joints which you now see has supervened. There is now also some muscular wasting, and stiffness of the tendons about the wrist. I suggest to you that the joint swelling is rheumatic and that the neuritis which preceded it is of the same nature. It should, however, be mentioned that the teeth and gums have been defective for many years. At the present time there is weakness and wasting of the muscles of the forearms, pain chiefly at night, and tenderness of the nerves and muscles. The skin of the hands is glossy. There is also some stiffness and swelling of the wrist-joints. Careful examination is necessary to reveal this and to distinguish this from the weakness and wasting of the muscles of the forearms. She is better, but has not made very rapid progress under anti-rheumatic remedies and galvanism and massage for the wasted muscles.

One of the worst cases of the same kind I have met with was that of a medical man, æt. 56 years, who consulted me some years back. He had been somewhat suddenly seized with acute pain in both arms a week or so before I saw him. The onset was attributed to a "chill" associated with indiscretions in diet. The pain, which was of a most severe kind, was attended by tenderness along the nerve trunks and acutely tender spots on the skin. There was at first no atrophy or paralysis, but the pain was increased by the slightest movement. The condition appeared to be of "gouty" origin, but in spite of strict dietetic measures, iodides, and anti-gouty remedies, it lasted for many weeks. The constant current administered by the double arm bath afforded only temporary relief, but counter-irritation in the shape of Paquelin's cautery seemed more efficacious. Many remedies were tried, and at length, in consultation with Dr. David Ferrier, it was decided to administer morphia hypodermically, and the patient then made more rapid progress. In the later stages notable wasting and paresis occurred in the forearms, and at this time electrical changes could be elicited, but once the pain subsided these gradually disappeared under electricity, and ultimately, though not until a year had elapsed, the patient completely recovered the use of his arms. The legs throughout were unaffected.

The lady whom you have seen presented some evidences of a generalised neuritis, but in the case just alluded to, the symptoms, which were much more acute, were strictly limited to the arms. In quite a number of cases I have failed after the most careful search to find any neuritis elsewhere,

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and I believe that brachial neuritis can, at any rate from a clinical standpoint, be regarded as a substantive affection, a clinical entity.

I have notes of some thirty cases of brachial neuritis. In addition to pain and tenderness of various degrees, weakness and more or less wasting, various skin phenomena may be observed. Small areas may be hyperæsthetic to light touch, or numb and anæsthetic. Very soon after the onset the skin of the fingers may become smooth and glossy, flushing, pallor, and, in rare cases, herpetic vesicles may be seen. It is only rarely that an electrical examination is justified, though sometimes, as in another case to be mentioned later, electrical changes can be detected. The course of brachial neuritis is essentially chronic throughout, or soon tends to become so. If untreated it may go on indefinitely. In two cases which I have observed over a long period of time (three and five years respectively) the pain spontaneously became gradually less and the muscular wasting seemed to come to a standstill. Sometimes other nerves become progressively involved. But, on the whole, the tendency is towards ultimate recovery, especially if the cause can be discovered and eradicated.

Brachial neuritis may require to be diagnosed from several other conditions. In occupation neurosis only one particular movement in one hand is involved. In brachial neuralgia there is pain with hyperæsthetic spots without any marked tenderness of the nerve-trunks and no anæsthesia or muscular involvement. I am able to show you examples of two other rarer conditions which may be mistaken for brachial neuritis. This man, æt. 42 years, came to the Hospital on the tenth of this month. Four years ago he first noticed twitching and weakness in the right finger and thumb; this was soon followed by wasting and weakness of the muscles, first in the right, afterwards in the left hand. It was not until two years later that the legs became weak. You will see that at the present time he has considerable wasting—the flaccid atrophic paralysis characteristic of lower motor neuron lesions—of the muscles of all four extremities, predominating in the hands and feet. The case conforms to the usual type of progressive muscular atrophy, but for six months the symptoms were localised in the right hand, and for two years they were limited to the hands only. He has, however, had no sensory changes and no pain or tenderness in the nerves such as almost invariably occurs in brachial neuritis, because the lesion, which is the same as that of infantile paralysis (poliomyelitis acuta), is limited to the anterior horns of the spinal cord. Here is another patient, a woman, æt. 50 years, whose illness also began with weakness in the hands, in August, 1906. This was soon followed by stiffness and pain in the shoulder-joint, and at the present time, which is fifteen months after the onset, she has almost complete paralysis of both arms. The hands are in the claw-like position of progressive muscular atrophy. But you will observe that there is considerable stiffness of the muscles of the hands and arms, which has come on during the last six months, and that this is accompanied by markedly increased tendon-reflexes of all the arm muscles. Lately the legs have become stiff and the knee-jerks increased. The case, in short, is an example of Charcot's amyotrophic lateral sclerosis. The disease began with weakness of the hands without pain, and presented great difficulty in diagnosis until atrophy limited to the thenar and hypothenar muscles became very obvious—a limitation never observed in brachial neuritis. Syringomyelia may begin in the same way and give rise to the same difficulty in diagnosis.

The ætiology of brachial neuritis comes under two headings—Local and Constitutional. Injury of various kinds, wounds, bruises, muscular strains, fractures, dislocations, are revealed by the history. They usually single out one nerve, often the musculo spiral, and only involve one arm. Tumours, abscess, bone disease, false rib, spinal pachymeningitis (syphilitic, tuberculous, or other), and other spinal or meningeal conditions often give rise to brachial neuritis (unilateral or bilateral) by pressure on the spinal nerve roots long before their local symptoms become manifest. This man, æt. 48 years, came to the hospital for deafness and vertigo, but you will see that he has typical musculo-spiral paralysis of the right arm, which is due to a fracture of the humerus in a railway accident twenty-three years ago. Students of anatomy look upon the musculo-spiral nerve as somewhat difficult, but it is not so if you regard it as the muscular and cutaneous nerve to the extensor aspect of the entire limb. In this case the nerve was injured in its spiral course round the humerus, and there is complete muscular paralysis of all the

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extensors of the hand and forearm, including the supinator longus. There is no enlargement of the supinator when the patient attempts to flex the forearm upon the arm with the hand in a state of pronation. But the local cause of a neuritis is by no means always as marked as it is in this case; more often it does not declare itself until long after the symptoms of neuritis have appeared. During the past ten years I have had five or six babies sent up to the hospital with paralysis of one arm, supposed to be suffering from infantile paralysis. This is usually quite a legitimate, or shall I say excusable, diagnosis, for, as in this child, æt. 11 months, there has generally been atrophy of the deltoid and some of the other muscles of the arm. Now, anterior poliomyelitis is rare, though not altogether unknown at this age, but I would advise you to suspect some injury to the clavicle or coracoid process by a fall upon the shoulder—a condition which actually exists in this little patient. But the precise position of the fracture is very difficult to make out in these fat children, and although this case has been examined by Mr. H. Fairbank and other surgeons, and has been submitted to X-ray examination, we are not quite sure even yet whether it is the coracoid process or the clavicle which has been broken. This is another of the many instances where the neuritis is easier to make out than the local injury or disease which produces it.

The constitutional causes of brachial neuritis increase in number as the possible causes of peripheral neuritis become known. There is no doubt that quite a number of cases are due to some disordered or poisoned state of the blood, the localisation of the symptoms in the arm or arms being determined by relative over-use of these limbs or some other localising factor, such as the special affinity of a poison for some particular nerve or nerves, as in the case of lead.

This man, æt. 43 years, came to us in November, 1906, for numbness without pain in both arms, and inability to extend both hands. The onset had been unusually rapid, only five days before he came. Three weeks before he had been laid up with severe colic. This is another case of musculospiral paralysis, only it involves both arms, and it differs from the preceding case in that the supinator longus is preserved. It is a typical case of lead poisoning, and in the early stages of this condition the supinator longus generally is preserved. The reason of this is not always grasped; it is because these degenerative lesions attack the ends of the nerves first, and, therefore, the extensor muscles of the forearms go first. The nerve supply of the supinator longus is derived from the main trunk of the nerve much higher up, before it divides into radial and interosseous. If the case is seen early the neuritis may not have spread up as high as this branch. The constitutional treatment in this case has been very successful; it has consisted mainly of iodide of potassium and mistura alba. He has also had the constant current (applied by means of the double arm bath) and massage three times a week, which have undoubtedly helped to restore the nerve.

There are, however, many other causes of peripheral neuritis which have been revealed of late years, and unless these possible causes be remembered they may be overlooked. I have met with instances of brachial and other peripheral neuritis which have been produced by the following toxic causes: alcohol, tobacco, rheumatism and gout (in which there is not, as in my third case, joint lesions, as in my second, to guide one to a diagnosis), tuberculosis and syphilis (not by their local but by their toxic effects), lead, influenza, diphtheria and other specific fevers, gonorrhœa, pyorrhœa alveolaris, and intestinal sepsis. Cancer and leucocythæmia have been mentioned by other observers as producing neuritis by their toxic effects or by their direct infiltration of the nerve trunks. Among these various causes lead and diphtheria are the only ones, so far as I am aware, which may have a special selective action on one nerve. The others seem to affect all the nerves of the arms in greater or less degree.

The treatment of brachial neuritis is important. The first, and often the most difficult, step is to identify the toxic or other cause in operation, and to direct the constitutional treatment to its eradication. Sometimes this is merely a matter of conjecture, and quinine, arsenic, salicylates, iodides, diaphoretics, and diuretics may be tried in turn. The constitutional treatment of this malady is, however, in most cases by far the most important, and repeated investigation should be made until one of the causes just mentioned is identified. Local treatment is at best only palliative or restorative. Amongst the local measures during the sub-

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acute stage to relieve the pain, dry heat is amongst the most successful—*e. g.* cotton-wool roasted at the fire and frequently applied, or a hot-water rubber bottle; dry heat- and light-baths act in the same way. Moist heat is sometimes successful in the form of arm baths or Turkish baths. Belladonna and opium, and other sedatives, may also be applied locally. Phenacetin is one of the most useful analgesics in these cases, and you will remember that in one of my cases morphia, under due precautions, appeared to act as a curative agent, as well as an analgesic. Galvanism is sometimes efficient in the same way, and it also preserves and restores the integrity of the nerves. Another useful agent for pain and restoration consists of counter-irritants over the nerve trunks chiefly involved in the form of small blisters or cautery. The use of Paquelin's cautery rapidly dotted along the course of the principal nerves once daily has several times relieved the pain and promoted recovery when other measures have failed. Nerve stretching and section of the corresponding spinal roots should only be resorted to after every other measure has failed.

Occupation Neuroses.

The occupation neuroses—writer's, telegraphist's, pianist's, violinist's, seamstress' and tailor's cramp, filer's, paper-glazer's, draper's, gold-polisher's palsy, and the like—have always appeared to me to be kindred affections to brachial neuritis. And though I admit that neuro-muscular exhaustion plays the leading *rôle* in the ætiology, I still believe (as I have already pointed out*) that a toxæmia is very frequently a contributory cause, and that in most cases constitutional treatment must be combined with the local treatment to effect a cure. There are six symptoms which may be met with in writer's cramp, which in my experience are, in order of frequency, as follows: (1) stiffness, cramp, or spasm, and (2) pain, sometimes accompanied by (3) muscular hypertrophy; (4) tremor, or (5) paresis, sometimes accompanied by (6) muscular wasting. My belief is that the last three are more often associated with a toxæmia than the first three symptoms; I have elsewhere recorded instances.†

Here is a classical type of multiple occupation neurosis. This woman, Mrs. R—, æt. 43 years, was first of all attacked with writer's palsy in January 1905, so she ceased writing with the right hand and learned to use the left, which soon began to tire if she used it much. Next she found it impossible to sew because of the "weakness" which came on in the thumb and index finger of the right hand. From her description the difficulty appeared to consist of incoordination and weakness in the thumb and finger. Later she lost the faculty of painting in the same way. Finally she found herself unable to garden because things fell out of her hands and because of the pains which now occurred in both arms. The prolonged cessation of these several movements did not effect a cure, and Dr. G. S. Leggatt, of Selhurst, sent her to this hospital. She attended from January to June of this year (1907), and to her great delight recovered the use of all the lost faculties, excepting writing, which she still preferred to do with her left hand. It is true she had galvanism from time to time, but I attribute her recovery largely to the administration of small doses of salicylates, aspirin, and remedies directed to gastro-intestinal derangement, because whenever she neglected these she relapsed. In June she was quite well and ceased all kinds of treatment, and tried to make up for lost time with her painting. She returned here on October 10th after an interval of four months without medicines, saying she had been working at a picture, but could now only with difficulty paint for half an hour at a time. This is a bad case which needs much more prolonged treatment, and I enjoined her to resume her old medicines without local treatment (or adequate rest, which she could not at the moment accomplish), and she has already slightly improved. I should like you to notice how one after another the faculty of writing, sewing, and painting rapidly became involved; this in itself points to the existence of some cause continuously in operation, as well as over-use, which only acted as the determining cause.

In the treatment the first essential is a temporary cessation of the offending movement. And this should be combined with local treatment, such as galvanism, systematic resistance movements, and massage. These are the most successful methods, in my experience, but Nussbaum's bracelet and other apparatus are favourably spoken of. Re-education of the movement in question, so as to correct faulty methods of holding the pen or

* 'Clinical Journal,' November 21st, 1900, pp. 72–75.
† *Loc. cit.*

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other instrument, is, where possible, also a most important matter. The patient should be taught to hold his pen *loosely* between his first and second fingers.

But in quite a number of cases success will not be achieved unless we can identify and eradicate the toxæmia or disturbed metabolism which is in operation. Many able observers* regard occupation cramps simply as exhaustion-neurosis of central origin. Undoubtedly this is to a large extent true, and particularly an exhaustion of the coordinating centres. Others regard them as a defective education, or shall we say a clumsiness, on the part of the operator—a view which is, in many cases, perfectly true. Others, again, regard them simply as a prolonged forced functioning of certain of the muscles and nerves of the limb. The possibility of a contributory toxæmia does not seem to have been sufficiently considered. But how otherwise, in the first place, are we to explain the fact that in some cases, as in the case about to be mentioned, careful inquiry reveals pain or other symptoms in both arms, and even elsewhere? Secondly, careful investigation also reveals, though I admit that this is somewhat exceptional, that electrical changes are found in one and occasionally in both arms. Thirdly, it has long been recognised in writer's cramp that after learning to use the left hand the left hand frequently becomes affected in the same way, in spite of every precaution; and, as we have seen, one after another kind of movement may be involved. The fact that out of 1000 clerks or telegraphists employing exactly the same movements perhaps only five or six will be troubled with cramp, may be partly explained by a faulty education or method of using the muscles. But the first three facts just mentioned undoubtedly support the view that in many cases there is a contributory causal toxæmia in operation.

There is also a fourth and most important fact supporting this view, namely, that the discovery and eradication of the toxæmia in operation promptly cures the disease. Quite recently a gentleman, æt. 52 years, consulted me for what he regarded as writer's cramp, which had been coming on gradually for about eight months, owing, he said, to the excessive amount of writing he had been called upon to perform. It is true that his writing was affected, as may be seen from the specimen I show you, the hand muscles going into a painful cramp as soon as he started writing, and that he had to give up his work on this account. But examination showed that the grasp of both hands was weak, that there was tenderness of the nerves and muscles on both sides, that the Faradic reaction was diminished, and that A. C. C. was greater than K. C. C. in both forearms. The case was one of bilateral brachial neuritis, which was rather more marked on the right side, and which had made itself manifest to him as a disorder of the faculty of writing. At first I was puzzled to account for bilateral neuritis, but complete examination revealed cardiac dilatation and irregularity, pale and sharp-margined discs, "nervousness," and slight muscular tremor—all pointing to some generalised toxæmia. The knee-jerks also were diminished, and he complained of being a trifle "shaky" on his legs, so the nerves of the legs were probably also becoming affected. I also learnt he had been smoking five ounces of tobacco per week, which he rolled into cigarettes and inhaled all day long. He at once knocked off the cigarettes and strictly limited himself to two pipes *per diem*. He was further treated by galvanism, massage, and systematised resistance movements applied to the arms, and he made a complete recovery from all symptoms in the arms, legs, and cardio-vascular system in the course of two months.

Very thorough investigation may be necessary to identify the toxin in operation: I have met with instances in which rheumatism, gout, influenza, pyorrhœa alveolaris, intestinal sepsis, alcohol, tobacco and lead have acted as contributory causes of writer's cramp or other occupation neuroses. I regret to say it has not always been possible in out-patient practice to sufficiently investigate and identify the contributory constitutional or hæmic defect in every case. We get a large number of cases of writer's cramp and other occupation neuroses at this hospital—particularly writer's or telegraphist's cramp or palsy from the post office—and many of these find it impossible to devote the necessary time to the electrical or other local treatment. But they improve under constitutional and dietetic treatment even while continuing their work, modified, perhaps, as far as circumstances will allow. There are two measures which I have sometimes found successful empirically in such cases: (1) the total prohibition of sugar, alcohol and meat from the dietary, and (2) the administration of salicylates, combined sometimes with aperients, over a long period of time.

December 9th, 1907.

* *E.g.* Professor Oppenheim, 'Diseases of the Nervous System,' Mayen's translation, London, 1904, p. 857.

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THE POSITION, DEFORMITIES, AND INJURIES OF THE FOOT DUE TO HIGH HEELS.

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Lecture III.

The female foot, which has been adapted to the requirements of high heels, is worthy of separate consideration, as its anatomy and physiology differ very markedly from those of the ordinary savage or male foot. It is a marvellous product in combining within itself the position of activity at the ankle-joint with raised heel, the abducted position of inactivity at the mid-tarsal joint, an arched instep, and deformities derived from positions both of activity and inactivity at the various phalangeal joints. Thus the female foot, which has been adapted to high heels, presents a curious combination of the various propositions set forth in the two previous lectures.

Observations on the position of the female foot whilst walking are easily made in any street. In the country, and particularly amongst the lower orders in towns, the subjects, if they are not weakly, walk with their feet straight forward or with the toes a little out. In the town or amongst the upper class they walk with their toes turned out so that their feet fairly constantly form an angle of about 35° or 40° with the direction in which they are walking. The usual explanation for this peculiar gait has been given in the small degree of genu valgum which is physiological to their sex on account of the greater breadth of the pelvis, their peculiar adaptation for childbearing. But this does not account for the differences in the gaits of the classes of women referred to above, as both bear children. It would seem that the essential differences lie in the heels of the footgear and the development of the tibialis posticus, the vigorous country woman having a low flat heel, and the upper class town woman a high and forwardly directed heel. This high heel places the foot in a position of activity or equinus at the ankle-joint, which position becomes fixed in the course of time by the adaptation of the structures to it. So that it becomes more or less impossible to extend, that is, dorsally flex the ankle-joint beyond a right angle, thus causing the possessor to lose a lot of power for activity, such as when springing. In the first lecture a good deal has been said of the disadvantage of this right angle talipes; and so it is to those who do not wear high heels. To those who do, the disadvantage is much less, as the foot is maintained a little plantar flexed by the height of the heels, increasing the extent to which extension or dorsal flexion can be carried from that position.

The high-heeled boot has another effect. Anyone standing with a raised heel is less steady than

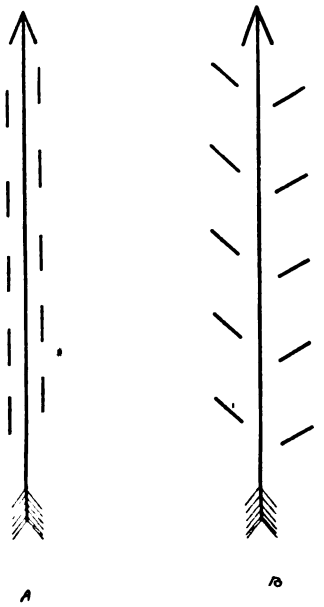

Fig. 1.—The arrow shows the direction in which the person is walking, and the lateral markings show the direction of the long axis of the feet. A. With active adducted feet. B. With inactive abducted feet.

when the heel is on the ground. To overcome this unsteadiness the calf muscles, other than the tibialis posticus, which maintain the "balance" of the tarsal joints, have to do a great deal more work, which leads to their greater development. This is somewhat strikingly confirmed by the greater rounded fulness of the calf muscles in those who wear high heeled footgear, in comparison with the comparatively flat calves so often seen in the lower classes, the heels of whose boots and shoes are not high.

But the high heel does much to replace the action of the tibialis posticus, the main-spring which has been shown to support and "wind up"

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the arch of the foot. Yet it is an everyday observation that women have arched insteps in spite of the deficiency of this muscle. Indeed, a high instep is a beauty which is sedulously cultivated by them. It is not acquired physiologically by developing the muscle fibres of the tibialis posticus, which is in fact poorly developed as compared with the other calf muscles. But the arch of a woman's foot is made and maintained to a large extent by the high heel mechanically causing plantar flexion at the mid-tarsal joint. The arch in a woman's foot is quite different to that of a man's or when low heels are worn. In the former the greater part of it is a physical, mechanical, and passive production, whilst in the latter it is a physiological, muscular,

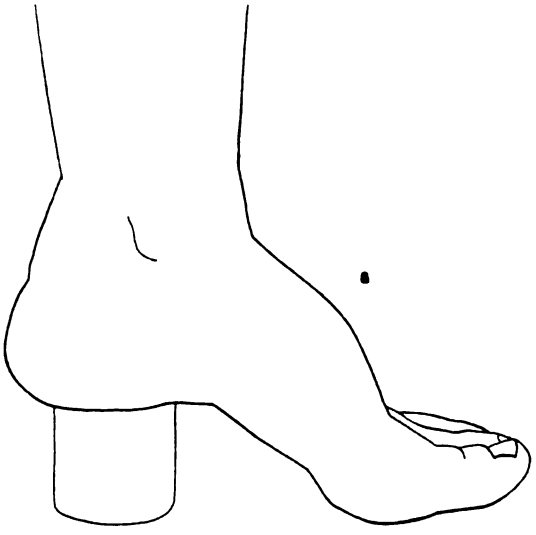

FIG. 2.—To show how the high heel mechanically causes plantar flexion at the mid-tarsal joint and arches the instep.

and actively maintained condition. The development of the tibialis posticus will vary in the two sexes. The arch of the female foot must not be conceived only in the light of the teaching of the two previous lectures, as to a great extent it is a purely passively made and maintained arrangement.

In the previous lectures it has been pointed out over and over again that with raised heels and arched feet the toes are directed straight forward or slightly inward. How is it then, that women who have raised heels and arched insteps turn their toes out so markedly? It has been shown that those who turn their toes in have adducted feet, and those who turn them out have abducted feet. And it is in this movement of the tarsal joints that the true reason why women turn their toes out is to be found. There is an interesting fact correlated with this. The skin under the ball of the big toe is "soft and silky," evidently sustaining no pressure, whilst under the third toe, or perhaps the fourth, is a callosity, sometimes two, which is sometimes a corn. The position of this callosity is also a sign of the abducted position.* Now, if from want of muscular development, or from one which is inadequate for the amount of standing to be done, or from weakly general health, the calf muscles yield, then the patient complains of pain and all the other symptoms of flat foot—pes planus. Yet the foot is not flat; it has an arched instep. One reason for this anomaly

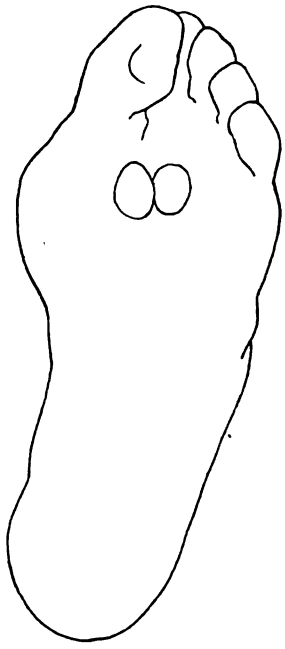

FIG. 3.—Drawing of the sole of a woman's foot accustomed to high heels. The callosity is under the third toe; in this case it consists of two parts corresponding to the heads of the second and third metatarsal bones.

has been partly explained in a previous lecture (CLINICAL JOURNAL, August 29th, 1906, p. 314), as the arch which remains after a certain amount has been removed by the abduction of the foot, which has assumed the resting position. Thus if a foot has a natural arch of the amount x, and the movement of abduction removes the amount y, then, when abducted, the foot will retain an arch represented by $x—y$. If x is greater than y, the instep

* CLINICAL JOURNAL, August 29th, 1906, Fig. 6, p. 314.

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remains arched as in the case of the women referred to above. But if y equals or is greater than x, the foot is truly flat. There is scarcely a better illustration of the use of the words "pes planus" in a foolish sense as when they are applied to the women above mentioned who have all the symptoms, but whose feet are not flat. Therefore I have suggested the deletion of the terms "pes planus" and "cavus" from the surgical nomenclature, and the substitution for them of "pes abductus" and "pes adductus" (CLINICAL JOURNAL, August 29th, 1906, p. 315).

The result of the present confusion of terms is that certain cases of abducted foot have been wrongly diagnosed, and, therefore, in all probability wrongly treated in consequence. As examples of

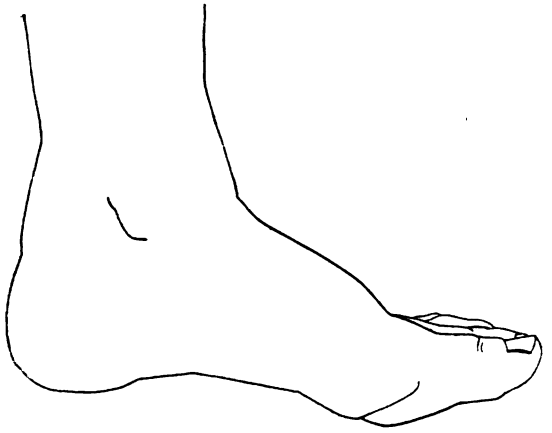

FIG. 4.—Sketch of the side view of a foot accustomed to high heels.

this I have already referred to the female cases, particularly those who wear high heels, and I would add to them cases after rheumatism, gonorrhœal and otherwise, and also after injuries such as Pott's fracture, oblique fractures of the tibia and fibula, fractures of the tarsus or metatarsus. In these, and as the direct result of the injury, the foot becomes abducted while its arching is not completely undone. The foot becomes flat in both the technical and symptomatic senses of the word, whilst in fact it is not flat. These unfortunate sufferers are only too often condemned as neurotic or malingerers through the lack of knowledge on the part of those who advise the judges or employers. The introduction of the use of the term "pes abductus" should prevent this.

The arch of women's feet is, therefore, of double origin, and is not entirely dependent on what has been demonstrated above as x—y. It has a greater and a purely mechanical causation in the high heel and the consequent passive plantar flexion of the foot at the mid-tarsal joint. So long as the high heel is present no amount of abduction can abolish the arch of the foot.

So far we have seen that the feet of those who wear high heels are associated with some talipes equinus at the ankle-joint and with some talipes

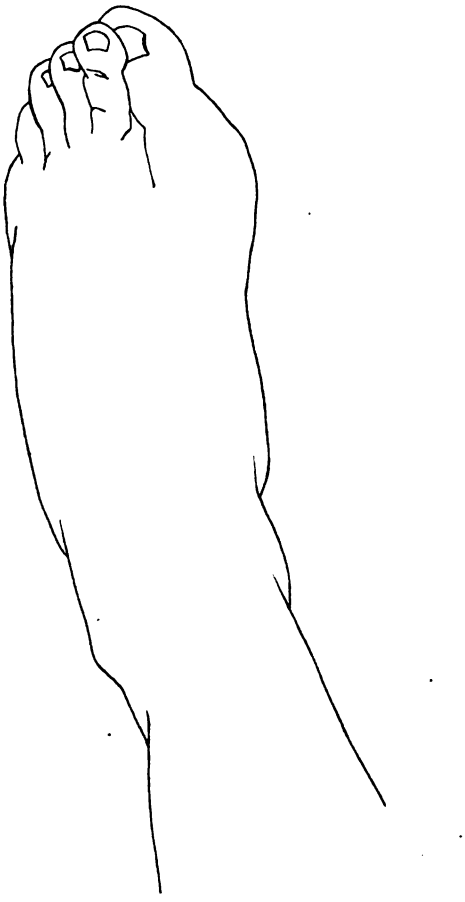

FIG. 5.—Sketch of dorsum of a foot accustomed to high heels.

valgus and abductus at the mid-tarsal joint. But when we come to consider the conditions and deformities at the metatarso-phalangeal joints we come again to apparent contradictions—deformities associated with both the active and inactive positions of the foot—because the high heels have endowed the abducted feet with an arch and a position of apparent activity. To sum up, the feet of those who wear high heels are in a position of activity at the ankle-joint, in a position of nactivity at the

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mid-tarsal joint, and have positions of both activity and inactivity at the various phalangeal joints. These last positions will be considered in detail.

As regards the big toe, it is commonly deficient in plantar flexion; that is to say, it may be a *hallux rigidus* of the active position (CLINICAL JOURNAL, September 5th, 1906, Fig. 13, p. 331, and Fig. 4 in the first lecture). Or this tendency may become exaggerated to form a *hallux extensus* of the active position (*loc. cit.*, Fig. 9, p. 329, and Fig. 5 in the first lecture). If reference is made to Fig. 2 it will be seen that a high heel mechanically produces this position of the great toe. Further, there is some *hallux valgus* which has resulted from the development of a bony boss on the inner (tibial) and plantar surface of the head of the first metatarsal bone, and is associated with the inactive position of the foot. This valgus position is not always the result of the shape and pointing of the boot as it is popularly supposed to be, because,

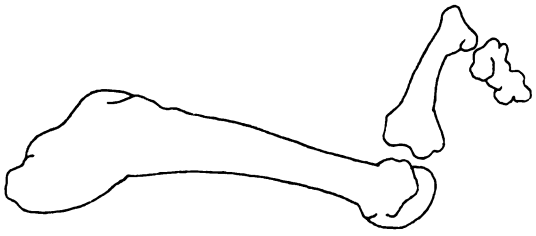

FIG. 6.—Minimus digitus extensus.

distal to the head of the first metatarsal bone the *big toe is not in contact with the upper of the boot.* The valgus position is, therefore, the result of the development of the inactive and abducted position of the foot.

At its interphalangeal joint there is commonly some *digitus flexus of the hallux* (Fig. 9 in the second lecture), which has been discussed in the previous lecture. But sometimes there is the opposite inactive deformity, the *digitus extensus of the hallux* (Fig. 7 in the second lecture). But this is not all; it is by no means infrequent to find that there is some valgus deformity at this joint, which is comparable in its pathology to the hallux valgus at the metatarso-phalangeal joint. It should be called *digitus valgus of the hallux* (Fig. 11 in the second lecture.)

The toes are in a position of extension (dorsal flexion) at the metatarso-phalangeal joints, and flexion (plantar) at the proximal interphalangeal joints. They are termed *digiti extensi*, and in the first toe it is usually called a hammer-toe (CLINICAL JOURNAL, September 5th, 1906, Fig. 17, p. 333). In the feet of women who have ceased growth, and sometimes when younger, the operative treatment of this hammer-toe by the ordinary means—excision of the head of the proximal phalanx—is frequently unsatisfactory. Amputation is the only satisfactory measure in those who have troublesome hammer-toes and will wear very high heels or whose feet have long been outraged by them. Thus I would put the treatment of hammer-toes in these cases upon a very different basis from that in the ordinary or male feet.

The deformation of the little toe is also worthy of special remark. It is flexed at the interphalangeal joints, and extended (dorsally flexed) at the metacarpo-phalangeal joint. But at the same time the proximal phalanx is drawn on to the dorsum of the head of the metatarsal, so that the toe scarcely touches or does not touch the sole of the boot (Fig. 10 in the first lecture). The position is an

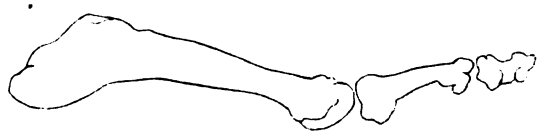

FIG. 7.—Minimus digitus rigidus (or flexus).

exaggeration of digitus extensus and may be called *digitus minimus extensus.* On the outer (fibular) side and over the head of the proximal phalanx is a corn, often very painful, which marks the point of pressure of the upper of the boot on the toe. The position of the toe is a sign of the active position of the foot, and the toe develops the corn as the result of an actively shaped foot taking up an abducted position. In inactive feet the little toe is not curled up so much as this and there is no corn. Though a little extended (dorsally flexed) at the metatarso-phalangeal joint, it is straighter, *minimus digitus, rigidus or flexus.* More than this, the axis of the little toe is not straight forward, but is directed forward and towards the mesial axis of the foot, *digitus minimus varus.* In the inactive position of the foot a varus deformity is usually seen, similar but less in degree, much more rarely a *digitus minimus valgus.* Thus the little toe becomes drawn into a position on to the dorsum of its metatarsal where it is functionless, (Fig. 10 in Lecture I). In consequence, if it becomes a source of pain or disability, which cannot be easily allayed, the toe

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should be removed without hesitation at the metatarso-phalangeal joint. There is no loss to the patient as the head of the metatarsal bone is left behind, which is the only useful part of such a little toe.

There is a point of interest in the wear of the heels to the shoes of those who have them high. If the tibialis posticus muscle is well developed, by its overaction it will cause the outer side of the high heel to become worn away out of proportion to the rest of the heel. So that outer part must be replaced. In those who are less vigorous and in whom the muscle gives up its struggle to maintain the muscular arch of the instep, the ankle turns inward and is said to "grow in." This can be often seen

FIG. 8.—Minimus digitus varus.

in the rapidly growing girls who wear short skirts. In them the heel is sometimes worn down disproportionately on the inner side.

In between the classes of those in whom the tibialis posticus overacts or has ceased to act are the great bulk of ordinary women. In them, as the outwardly turned abducted foot is brought forward as in walking, the outer side of the heel is rubbed down. I once saw this point brought up very cleverly in a law court by the barrister representing an insurance company against a man claiming compensation for an abducted foot after a Pott's fracture. He argued that how could a man who had a flat foot, and who therefore walked on the inner side of the sole, wear away the outer side of his heel? The true answer lay in the possible overaction of the tibialis posticus and the method of advancing of the abducted foot. But without previous thought it was not easy to give an impromptu explanation from the witness-box.

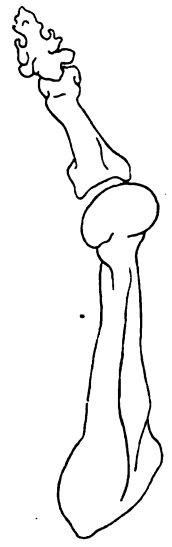

FIG. 9.—Minimus digitus valgus.

The introduction of the high heel usually, but not necessarily, forces the feet of those who wear them into a position of abduction. But at the same time it minimises to some extent the disadvantage of this by shortening the tread of the

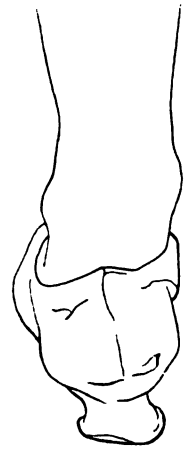

FIG. 10.—Back view of the left foot with wearing down of the outer side of the heel.

foot—that is to say, by approximating the heel and toes. This shortening of the foot also makes it appear smaller—an illusion much appreciated. When a person is running or jumping they move on their "toes." To extend this principle, the

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shorter or smaller the tread of the foot the greater the advantage in activity. Unintentionally, boot makers have aided in this by giving the high heel a direction forward. But unfortunately it produces movement of the upper of the boot on the tip of the heel where sometimes a very painful corn or blister arises, the blister if the trouble arises quickly, the corn if it comes slowly. Thus one of the troubles which may accrue from high heels is a sore place on the back and outer side of the heel which is difficult or impossible to cure so long as high heels are worn and the skin has not been allowed at first to quiet down, and, later, to harden and become unimpressionable to the friction.

Of the deformities associated with the use of high heels that of the "growing in" of the ankles is the most frequent. It is often seen in quickly growing girls. But as this "growing in" is usually found in young subjects and high heels are taken to at a somewhat later age, it is not always impossible to blame them alone. Combined with these ingrowing ankles it is common to find an outward curvature of the lower third of the tibia. The deformity which is next commonly associated with the use of high heels to the boots and shoes is genu valgum or knock knee. This exaggerates the slight degree of the deformity which is natural to every woman on account of the greater breadth of her pelvis. It is illustrated by the greater degree of the deformity present in the higher classes of civilised women as compared with savage women.

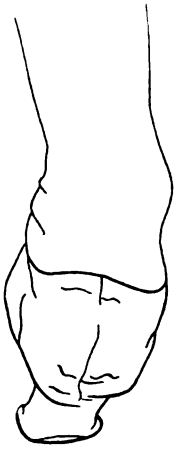

FIG. 11.—Back view of the left foot with wearing down of the inner side of the heel.

With regard to injuries, those who wear high heels very seldom injure the foot proper; but rather does the damage occur higher up. For instance, they commonly twist their ankles, less frequently so severely as to sprain them. This comparative immunity from injury is doubtless due in great part from the general infrequence of an injury to the female sex. But it is of great interest in view of the statement made in the second lecture, that abducted feet are more liable to injury than adducted. Those who wear high heels have also abducted feet, but the heels seem to confer safety on the bones and joints of the feet at the expense of an increase of comparatively trivial injuries higher up the leg, such as "twists" of the ankle.

To sum up, high heels make the foot appear smaller and neater than without them. Mechanically it gives the foot a high instep; it causes numerous deformities of the toes and, perhaps, painful callosities under the big toe, on the sole opposite the third toe, on the dorsum of the little toe, and often by the tip of the heel; they exaggerate certain conditions which are physiological to women, such as some degree of genu valgum. They seem to save the foot from severe accidents at the expense of a greater number of trivial ones. In addition, unfortunately, they weaken the tibialis posticus muscle and require greater development of the calf muscles than in those who wear low heels. Most women cannot meet this additional demand, with the result that the foot becomes abducted into the position of rest, which requires little or no muscular effort, becoming symptomatically but not technically "flat," whilst the ankle goes "off the straight," either "growing in or out."

FIG. 12.—High-heeled shoe, showing the forward direction and displacement of the heel.

December 9th, 1907.

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A CLINICAL LECTURE

ON

SCARLATINAL OTITIS.

Delivered at Monsall Hospital.

By A. KNYVETT GORDON, M.B., B.C., B.A.Cantab.,

Medical Superintendent of the Hospital; Lecturer on Infectious Diseases in the University of Manchester.

I PROPOSE to-day to show you two cases of infection of the middle ear arising in the course of an attack of scarlet fever, and I shall also make a few remarks on the pathology and treatment of scarlatinal otitis in general.

Let us first recall a point in the anatomy of the middle ear. If you look at these specimens of dried temporal bones, you will notice that the Eustachian tube enters the tympanic cavity about half way up on its anterior wall, and that if the tube could be prolonged across the tympanum it would hit the opening of the aditus, or passage leading to the mastoid antrum. In fact, in children under the age of ten, or thereabouts, the Eustachian tube, tympanic cavity, aditus and mastoid antrum are all in the same straight line. Here are specimens from children aged one, two, three, five, seven, and nine years respectively, and they all show this point well. The importance of this is that any infecting agent coming up the Eustachian tube finds it just as easy to get into the mastoid antrum as into the tympanum, while any bacteria entering from the external auditory meatus would have to go round the corner to reach the mastoid antrum. I may also remind you that Young and Milligan have shown that the Eustachian tube, tympanum, and mastoid antrum are developmentally a single straight tube, and also that if a perforation be made in the tympanic membrane, smoke or fluid passed up the Eustachian tube does not come out through the perforation until the mastoid antrum is full of it. The other points that these temporal bones illustrate are familiar to you from your anatomical work, and I need not allude further to them here. I would ask you, however, to note that in these young bones, while the mastoid process is very small, the mastoid antrum is comparatively large. Note also the varying position of the lateral sinus, and the thinness of the bone separating the attic and antrum from the middle fossa of the skull.

We will now see the first patient, who has a mild attack of scarlet fever which began ten days ago. He is a well-nourished boy, æt. 6 years, who is obviously not seriously ill. His skin is, as you see, peeling freely, and his fauces are somewhat injected; there is healing ulceration of each tonsil. He was admitted here on the fourth day of his illness, and his temperature fell to normal three days ago.

There is a fairly profuse purulent discharge, which is slightly fœtid, from his right ear. This is of four days' duration, and has never given him any inconvenience. He will tell us that his ear has never hurt him at all. In point of fact, the otorrhœa commenced when he was asleep, and some pus was noticed on his pillow the next morning.

When the discharge has been wiped away you will be able to examine the ear, and the first point to which I would direct your attention is a shining patch of bone slightly above the centre of the field of the otoscope. This is the promontory of the tympanum, and it covers the first turn of the cochlea. This gives you your bearings, and on looking round you will see that the tympanic membrane has practically disappeared, being represented only by a fringe. You are looking at a mass of granulation tissue covering the inner tympanic wall and surrounding the promontory, which has been denuded in patches of its muco-periosteum. The greater part of the ossicles lies above the field of view, but you will see a speck of bare bone hanging down, which is the tip of a carious malleus. The whole field is secreting pus; there is but slight impairment of hearing.

These are what may, I think, be described as fairly typical appearances in a case of acute scarlatinal otitis, and the first point to which I would call your attention is that they are what you would expect to see in a case of ordinary or catarrhal otitis, from cold, sea bathing, or what not, that has been discharging for several months, the duration of this being, as I said, four days only.

The main difference between the two conditions, however, is this: in scarlet fever you have inflammation of the whole tract, starting from the Eustachian tube, and in catarrhal otitis the lesion is

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confined to the lining membrane of the tympanum and the infection is from without, *viâ* the external meatus.

The origin of such a condition as this is to be found in the naso-pharynx, which, in the acute stage of scarlet fever, is always infected with micro-organisms, and not infrequently contains sloughing adenoid tissue as well. The spreading of the infection up the Eustachian tubes is, therefore, not to be wondered at, and it does, in point of fact, occur in about 20 per cent. of all cases. The onset is almost always painless, while any of you who have suffered from an attack of catarrhal otitis will remember that the pain was then very acute until the discharge appeared. In scarlatinal infection the perforation in the membrane is almost always a large one and the discharge copious. In catarrhal otitis the perforation is frequently small and badly placed from the point of view of drainage of the tympanic cavity.

This boy is quite comfortable and in no apparent danger. In all probability one of two things will happen to him: either the discharge will dry up gradually, after remaining profuse for a week or so, the granulation tissue in his tympanum will be absorbed, and the membrane will re-form, or be replaced by scar-tissue. Unless Eustachian obstruction supervenes there will probably be only slight impairment of hearing, which will itself disappear in a few months. Or the discharge may not dry up, but continue and become more fœtid, and, as we say, chronic. Whether this is taken seriously by the patient or his relatives depends in any given case very largely on their social position. If nothing is done, one of three events is probable: either general septicæmia from the infected ear, or intra-cranial infection, or extension to the lateral sinus with resulting pyæmia. Chronicity of ear-discharge in scarlet fever almost invariably means bone disease, and this is apt to occur at a much earlier period in scarlatinal than in catarrhal otitis; in fact, it is probable, from considerations with which I need not trouble you now, that we have in scarlatina an osteitis, wherever the tract is bony, from the first. This does not, of course, invariably go on to necrosis, but it is very apt to. As a matter of fact, intra-cranial or sinus extension in the acute stage of a scarlatinal otitis is not common, though I have seen both more than once, but septic absorption with resulting pyæmia from undetected caries is, unfortunately, by no means rare.

I think the second case may possibly illustrate this point more clearly. Here is a girl, æt. 7 years, who is obviously somewhat ill; her temperature is 101·5° F., and her pulse rate 130°. I may tell you that she has scarlet fever, which began a fortnight ago, three days before she came in here. Her throat was rather more severely affected than in the previous case, and the temperature did not fall to normal until four days ago, when it began to rise again in a manner not unlike that which we see in the case of enteric fever, ascending two degrees at night, and falling one degree in the morning. Yesterday, the right ear commenced to discharge again without any preliminary pain, and I found much the same appearances when I examined the tympanum as in the last case. The membrane was nearly all gone and the tympanum full of granulation tissue. When the discharge commenced, however, the temperature did not fall, and a careful examination failed to reveal any cause for this in any other organ but the ear. I cannot show you the ear now, as it has been prepared for operation.

Why is that? What indication have we that an operation is required? There is no pain, and the intra-tympanic appearances show nothing inconsistent with an ordinary attack of scarlatinal otitis, but the sister of the ward reported that the ear was tender when it was touched behind, and on examination I found slight, though well-marked tenderness on pressure over the mastoid process. That was early this morning, and at the same time the discharge was noticed to be much smaller in amount. So we have four signs. The patient is obviously worse—the pulse-rate tells us that. The discharge is less, the temperature is raised, and there is slight mastoid tenderness. There is, you will note, no obvious external sign of mastoid abscess, but you do not by any means frequently get local redness or swelling in scarlatina. Obvious mastoid abscesses with external signs are only seen in ·5 per cent. of all scarlatinal cases, while the number of cases in which carious bone is present in the mastoid process is much greater. I think it therefore probable that we shall find carious bone in the mastoid process of this patient. At all events, I am going to open the mastoid antrum now, and we will send the patient to the theatre at

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once, so that you may have an opportunity of seeing what the condition of things is.

I have now opened the mastoid antrum with the gouge, and you will note that pus is welling up in the wound. Its volume is not great, but it has evidently been imprisoned under considerable pressure. But this is not all; now that the mastoid antrum is freely opened and its cavity cleansed, you can see that the bone surrounding the lower part of the antrum is carious and dotted with little beads of pus and fragments of granulation tissue. I will remove all this until healthy bone is reached. You will now see that almost the entire mastoid process together with the cells at the root of the zygoma are infected and carious. There is in this case obviously no reason why we should touch the tympanic side at all: it will suffice to clear away all diseased tissue on the mastoid side of the posterior meatal wall, paying particular attention to the roof of the antrum and aditus, and we will leave the wound open for daily irrigation for the present.

I think you will all agree with me that the extent of the bony disease is greater than the slightness of the local symptoms would have led us to expect in this case. But this is in scarlatinal cases not uncommon for the reasons I have mentioned. (In this case the temperature fell to normal the next day, and the patient made an uneventful recovery.)

Turning now to the subject of scarlatinal otitis in general, what are we to do for our patients? Well, in the first place, we seldom have occasion to perforate the tympanic membrane, as we are advised to do in some of the text-books for the relief of pain, because this is generally conspicuous by its absence, and the natural perforation is usually quite sufficiently large for drainage. We can, and should irrigate the tympanic cavity thoroughly, preferably with a large volume of fluid at a low pressure. We will not use a syringe, as that generally delivers a small quantity with a varying, and usually excessive force. At first it is not necessary or advisable that the fluid should contain any strong chemicals. Sterile salt solution, or even tap water at a temperature of 100° F. will suffice. Later on, say after a week, some astringent such as sulphate of zinc, or carbolic lotion may be used. If the discharge is thick and offensive there is nothing so useful as the instillation of pure peroxide of hydrogen solution followed by irrigation with water. After this, more peroxide may be applied to the tympanum illuminated through the aural speculum with a cotton-tipped probe. Any large masses of secretion may be removed with forceps, but it is not advisable to take away granulations or even to apply caustics.

Under careful irrigation, the large majority of scarlatinal otorrhœas can be cured, but there is a residuum in which the discharge persists, and in these cases there is almost always some carious bone somewhere out of sight either in the attic or aditus or more commonly in the mastoid process. This will not "work out" of itself, and it must be removed by an operation.

The first step is to open the mastoid antrum so that that cavity and the aditus can be inspected and explored with the probe. Any carious bone should now be removed, and any sinuses opened up. These usually lead towards the tip of the mastoid or towards the groove for the lateral sinus.

When the mastoid side has been cleared of diseased bone the tympanic cavity should be thoroughly examined with the reflected light—this should be used for every mastoid operation—and any masses of granulation tissue or carious patches of bone removed with a small curette. Two courses are now open: we may either leave the ossicles untouched and rely on drainage through the posterior wound, which is left open to subsequently heal by granulation, or we may take away the ossicles and remove the posterior wall of the meatus, thus making one large bony cavity. The cartilaginous meatal wall is in this case split and used to partially cover the roof and floor of the resulting cavity, the posterior wound being sewn up. This is called the "radical mastoid operation," and the cavity is subsequently dressed through the meatus, the wound in the bone healing, not by becoming obliterated with granulation tissue, but by epithelialisation of its walls. It remains as a cavity.

Each case must be considered separately. No general rule can be laid down. Personally, I am inclined in hospital cases, where one is almost sure that the patients will not be taken to a medical man subsequently, however strongly one may advise the parents to do so, to prefer the radical operation. One point is worth noting, viz. that in scarlatinal cases the prospect as regards the hearing is very much better than in catarrhal cases. These latter are not as a rule subjected to the radical

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operation until several months or even years after the commencement of the otorrhœa, and in the scarlatinal cases I have been accustomed to operate generally from six weeks to three months after the onset of the otitis, in fact, as soon as I feel sure that there is carious bone somewhere out of sight and reach. I have never failed to find this carious bone.

I have got out the figures for 106 consecutive radical operations performed here, in each case for the cure of otorrhœa, a few cases in which the operation was done for local mastoid symptoms not being included, and I find that two died of meningitis from lymphatic metastasis, and in one other case the hearing was worse after the operation. In the remaining 103 the hearing was improved. Depending partly, but certainly not entirely, on the improvement of hearing is a marked alteration in the mental state of the patients after the operation; these patients are often stupid as well as deaf, and in almost every case the nurses have noticed that the child has been much brighter after the operation. I do not think it safe to treat these cases of scarlatinal otitis by any form of intra-tympanic ossiculectomy. The parts removed are not in view during the operation, and it is impossible to know in any given case whether the whole of the diseased bone has been taken away or not. A general anæsthetic is required (in children at all events) for the operation, and the duration of the after-treatment is as great as that for the radical operation. The risk of meningitis from incomplete removal of carious bone is also a factor to be dealt with.

I show you here a patient on whom the radical mastoid operation was performed a month ago. Though the only lesion seen with the speculum before operation was destruction of tympanic membrane with granulation tissue filling the tympanum, the presence of carious bone was suspected because on drying the tympanic cavity carefully with a cotton-tipped probe pus was seen, on careful inspection, to be coming down from the region of the attic. Moreover the discharge resisted irrigation for over a month, and it was slightly fœtid. At the operation a large hole was found in the roof of the mastoid antrum and tympanic attic, through which pus was draining (fortunately freely) into the tympanum—there was, in fact, an extradural abscess. The mastoid process was carious nearly to its tip, and there was a small erosion in the bony wall of the lateral sinus, with a patch of granulation tissue on the vein itself. You will now see that the cavity is almost entirely covered with epithelium and that there is no sign of carious bone anywhere. You will notice that with the other ear stopped up she can hear questions delivered in a low voice at a distance of six feet.

I think I have now said and shown enough to impress upon you the fact that the dangers about which you have learnt elsewhere to connect with the presence of otorrhœa are of very pressing importance when the otorrhœa is of scarlatinal origin.

December 9th, 1907.

Differential Diagnosis of Jaundice.—Arnsperger describes various forms of jaundice, and states that it is possible to differentiate the icterus from gall-stone occlusion from that due to a tumour or resulting from a parenchymatous affection of the liver. In case of gall-stones obstructing the common bile-duct, the jaundice commences suddenly, with pain, after colic with and without icterus. The patient shows signs of cholangitis and seems very sick, with sometimes a general septic condition. In cases of long standing the liver is enlarged, with a tendency to cirrhosis; in the early stages only the right lobe is enlarged. The gall-bladder can not be palpated, but the region is tender and also the epigastrium. The stools are sometimes with and sometimes without admixture of bile; the urine contains much bilirubin but no urobilin, and the blood is approximately normal. In case of occlusion of the common bile-duct by a malignant tumour, the jaundice comes on gradually and without pain, but with increasing cachexia. The liver is enlarged throughout. The gall-bladder is palpable and distended. Ascites is also observed, and the stools are constantly clay coloured. There is also a tendency to hæmorrhage and a low hæmoglobin percentage. Functional icterus from a parenchymatous affection of the liver comes on slowly, with remissions sometimes for years; pains at times, but no actual colic. Syphilis or chronic intoxications will be discovered in the anamnesis. The liver and spleen are both enlarged, but there is no tenderness in the gall-bladder region, and the gall-bladder cannot be palpated. —*Journ. A.M.A.*, vol. xlix, No. 20.

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THE CLINICAL JOURNAL,

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EDITED BY L. ELIOT CREASY.

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A CLINICAL LECTURE

ON

A CASE OF NEW GROWTH OF THE LUNG.

Delivered at St. Bartholomew's Hospital.

By NORMAN MOORE, M.D., F.R.C.P.,
Physician to the Hospital.

IT is my intention to lecture to-day on a case of new growth of the lung which is at present in Mark Ward. New growth of the lung, that is to say, primary new growth there, is not a very common condition. If you look at the hospital statistics you will see that there are very seldom as many as five cases of it in the hospital in a year, male and female. Of course, the appearance of new growth in the lung as a secondary situation is not very uncommon. In most cases of long-standing carcinoma in which the liver has many secondary growths, a few may be found on the surface of the lung, but those are scattered patches, which do not give rise to any symptoms, and are purely of pathological interest, as showing the direction in which secondary growths grow. You may occasionally see that in cases of primary new growth in the stomach where the liver is affected, and the lymphatics infected, that there are a few scattered patches on the surface of the lung. And you may see them in other varieties of abdominal new growth. Again, you see such secondary patches on or in the lung in cases of carcinoma of the œsophagus. If it has lasted long, and particularly if it is a large and not very dense growth, you nearly always find some such on the surface of the lung. But those growths, as I have said, are purely pathological; they have no clinical interest because they do not give rise to any symptoms and they could rarely be recognised during life. They do not cause extensive pleurisy. But the case I am going

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to show you to-day is of quite a different kind. It is a primary growth of the lung, and, as I have already said, such growths are rather uncommon. It is very important, and yet it is often extremely difficult, to recognise them when you come across them; and that is the reason I have chosen this comparatively rare case to-day for my lecture; it is a typical example of new growth of the lung. Now, as I say that, you may very properly ask am I absolutely certain of it? Is there any possibility of error? I could not answer that question in the affirmative; there is a possibility of error. That possibility will always exist where you cannot actually see the growth and where you cannot recognise anywhere on the surface of the patient's body a secondary growth. Therefore there is a possibility of error, and it is right to put that before you, because that is one of the difficulties of this particular morbid condition that you very often cannot arrive at absolute certainty about it. Nevertheless, I believe that this is a case of new growth of the lung. I will go further than that and say I believe it is a case of new growth affecting chiefly the left lung. I will examine the patient before you. First, we must find out what the physical signs are. He has something wrong with his chest: we always try to discover what physical signs there are in any morbid condition of a patient's chest. He is a carman, æt. 47 years. You can see that he is rather thin and that his chest is a little rounded, which would suggest to one that there was some emphysema present, but it is probably a long standing condition. You can observe there is very little difference between the condition on full inspiration and that on expiration; the costal angle as he breathes is hardly moved at all. Those are generally the signs of there being some emphysema of the lung, a condition preceding the disease from which he is now suffering. That is what we observe on inspection. I get him to speak while I have my hand on his chest, that is to say I practise palpation. I feel perfectly distinct vocal vibrations on the right side, but no vocal vibrations on the left side. Even quite up under the clavicle I cannot feel any vocal vibration. That, of course, is rather an uncommon physical sign. If you will remember the cases which you have seen in the wards I am sure you will recall that it is rare to find total absence of vibration right up to the clavicle on either side. It is much commoner to discover such absence behind at the base, but rare to find it quite up to the clavicle. We know many ways in which it may be produced, but, on the whole, it is a rare thing. Now we will percuss his chest. The right side is perfectly resonant, as you will hear even from a distance, but the left side is absolutely dull, even wooden. I will now percuss in the axilla. In the whole of the left axilla dulness is also absolute, whereas on the right side resonance is present. Notice that he can sit up without obvious distress. There is absolute dulness on the left side from the back and there is absolute absence of vocal vibration on the left side. At the apex I can hear faint breathing, and all over the back I can hear a very faint distant respiratory sound. On the right side the breathing is natural. There are places quite low down where I cannot hear anything at all, but over most of the back I can hear a very distant respiratory sound. In front I hear no breathing sound at all below the clavicle; above I can hear a faint sound. The right side is natural. The heart sounds are quite natural and the heart is very slightly displaced to the right. He says he feels easy now; he has been extremely short of breath. Those are the points to observe, then: that he has absolute stone-like dulness on the left side, and almost complete absence of breathing, very faint and distant breathing sounds to be heard behind. A day or two ago when we examined his chest there was a very curious note to be heard high up, but it has now disappeared. There was a hyper-resonant, tympanitic note just at about the second and third ribs. In a garden of mine in the country I noticed a curious resonance on percussing with a spade the grass under a cherry tree at the edge of a bowling green. I got the gardener to dig down expecting to find a hollow of some kind in the ground. Digging showed that there was no large cavity, but that under a long, thick old root of the cherry tree the soil was much looser than elsewhere, and had sunk from the root for a few inches, leaving a hollow. The gardener had evidently expected something more. The note reminded me of that hyper-resonance in this man's chest, and some similar condition of sub-structure may explain it such as a mass of growth extending to a bronchus.

Let us now discuss the absolute absence of vocal vibration and of breath sounds. What do those point to? You have all seen a common condition

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in which they occur, namely, pleural effusion. When one side of a man's chest is full of fluid, whether it is serum or pus, there are no vocal vibrations; any breathing sounds are very remote indeed, and they may be altogether absent. There is absolute dulness here, so we have here the physical signs of pleural effusion. But before we proceed to interpret those signs I must read you the history of the case. He was admitted on August 28th and he then had extreme shortness of breath. He himself says he was quite well on July 22nd. He is able to fix that date, and soon after that he had pain across the front of his chest, worse on taking a breath, and a day or two later he had distinct shortness of breath, he sweated very much, the pain continued, and the dyspnœa persisted and increased. Both were worse at night and he steadily got worse until his admission, which was about a month after the occurrence of the first symptoms. He had lost flesh, and occasionally, besides the pain in the chest, he had pain in the left axilla and left upper arm. He had had no serious previous illnesses. When he came in he was somewhat emaciated, and he has become more so since. He looked very ill, which is an important point in relation to the diagnosis, and he showed great distress in his face. He also had orthopnœa. The respirations were very shallow and rapid, about 48, and at times they were gasping. The left side of his chest looked as if it bulged a little, but it was fixed and it was dull to the apex. It moved a very little just now, back and front. There was a sense of absolute resistance on percussion, and no vocal vibrations. The right side was normal. I have examined him minutely twice and his physical signs have been gone into on several occasions. What I have shown you here just now, and what I have read to you from the note, have on every occasion been the physical signs. The only differences are very slight ones, and I will mention them. One is that now and then very faint and distant breathing has been heard, but that has only been after fluid has been let out from the chest. I will enter into the question of fluid soon. And secondly, on his left chest, where I pointed out to you, near the second rib, there has been a peculiar tympanitic note. It is quite absent to-day, but the first time I examined him it was extremely plain and the second time it was plain, though it was audible for a smaller area than before. Such was his state when a needle was put in and some blood-stained fluid came out. And the next day four pints of deeply blood-stained fluid were aspirated. Here is a test tube containing some of the fluid taken at the last tapping, and you will see how deeply blood-stained it is. What came out was not a mere teaspoonful, but four pints of fluid, all quite as deeply stained as that. Here is a tracing showing the form of the chest, and the relative sizes before and after tapping; there is a difference of nearly one inch diameter on the two occasions. He was rather better after he had been tapped on August 29th, and on September 10th he was again distressed and two pints of the same kind of fluid were let out. On September 21st he was again aspirated and five and a half pints of similar fluid were withdrawn. Then again on October 7th about the same quantity was withdrawn. The fluid was examined in the Pathological Laboratory. One tube was found to be sterile. The other, on the third day after shaking, grew a white staphylococcus, probably a contamination product, and the same thing was found on another tapping. He coughs up a little every day of thickish fluid, and this has been examined for tubercle bacilli but none have been found. The physical signs, as you saw, were those of pleural effusion. I want you to notice that, because it is not invariably the case in new growth of the lung. Sometimes there are different physical signs, but on the whole it is the commoner condition; the physical signs were those of pleural effusion. But how does the case differ from one of pleural effusion? Why do not we think it is simply a case of chronic pleurisy? First of all, the character of the effusion: it is deeply blood-stained. That is a very rare condition unless there is a new growth of the lung. When I use the word "lung" I am including in it the whole of the respiratory organ from the pleura to the bronchi. The terms that we use in speaking of lung diseases are a little misleading. We speak of bronchitis, but who can separate the small bronchi from the lung? Who can say that in a disease of the air vesicles small bronchi are not also affected? It is clear that that is only a very rough term, and must not be used with any exact anatomical meaning. The real fact is that that word bronchitis, which was introduced into medicine about the year 1808 (by

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Dr. Charles Badham, in his 'Observations on the Inflammatory Affections of the Mucous Membrane of the Bronchiæ'), owes its prominence and its constant use to the fact that the general public would rather not think that their lungs are affected, and so they like to hear that it is only the bronchi which are involved. But the only true anatomical way to look at the lung is as a structure consisting of everything contained within the pleura, and it is possible that this growth may have involved a very large area of pleura. If it had involved even a small one it would have been sure to have reached the pleura at some point. Therefore blood in the effusion which is let out will always suggest to you the presence of new growth. Is it an absolute sign? If it were there would be no need to take those precautions with regard to certainty of diagnosis. It is not quite an absolute sign. In thinking of these things it is useful to look back upon one's own experience. By that I mean not only one's own observation but the effect upon one's mind. And I can remember a period in my own experience in this hospital when I thought that if you let out deeply blood-stained fluid you always had to do with a new growth; and I remember quite well a case which impressed upon me the fact that that is not absolutely true. It was a man in John Ward who had a very large pleural effusion and he was about 38 years of age. The effusion was tapped and it was deeply blood-stained. It was tapped a second time and was blood-stained. But the man after many weeks got quite well, no further effusion appeared, his lung expanded, and he left the hospital quite well. It was clear that it was not a case of new growth. Since that I have seen a few examples of deeply blood-stained effusion which were not associated with new growth, but such cases are very, very rare. Please be quite clear in your minds that I mean an effusion like that being passed round in the test tube. You have to bear in mind that the mere act of paracentesis may cause a slight hæmorrhage, and so the fluid may be tinged with blood, but that is a different thing from what you see in that tube, for the whole fluid is a dark red—the fluid is profoundly blood-stained. One difference, therefore, between this case and an ordinary case of pleurisy is in the nature of the fluid. It is not clear serum, it is not pus, but it is a deeply blood-stained fluid. Is there any other difference? Yes, there is a difference in the physical signs. The dulness which you get when the chest is full of fluid is often very complete; there is not the faintest trace of resonance in it, but it is seldom quite complete a few hours after you have let out, say, five pints of fluid. Nearly always some resonance has returned in those circumstances. In this case that wooden resistance is to be felt almost all over the left side of the chest after five pints of fluid had been taken out, just as it was before that procedure. You can see many explanations of that. But that is characteristic of a large effusion due to new growth, as distinct from a simple pleurisy of any kind. Another strong point in favour of growth is the fact that each time that this fluid is let out it has exactly the same characters; it is always this deep blood-stained fluid. Are there any other signs? You will remember I mentioned that there was a very curious tympanitic note to be heard near the second rib in front. I looked up the notes of a few cases of new growth of the lung that I have observed myself. One of them was that of a man, æt. 37 years, who had a large new growth which affected one of his lungs, though I believe it was primary in his pericardium. It became a growth of the lung and extended directly from the pericardium into the lung; and I found these physical signs recorded. It occurred twenty-five years ago. My note says: Absolute dulness on the left back from the spine of the scapula downwards. In front, a peculiar hyper-resonant note just below the left clavicle. Elsewhere on the left side in front, absolute dulness. Right side of the chest normal.

At the post-mortem there was found an extremely dense growth beginning in the pericardium and extending into the left lung and occupying the whole of the upper part of the chest, pressing on the bronchus. There was a sound exactly like that to be heard in this patient, and it has been described by other observers. So that although that is not a sign which proves the presence of a growth, it is at least a sign which has occurred where the growth has pressed upon the root of the left lung. And therefore I added to the notes that there are physical signs which make it almost certain that that patient has a new growth affecting the left lung.

Now let us turn from the physical signs to the symptoms. Do those symptoms support the view that he has a new growth? They came on very

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suddenly; he knows he was quite well on July 22nd, but before the end of that week he was very ill. He had no particular feverishness, and he has had only a very slight rise of temperature since he has been in the hospital. Is that characteristic of new growth, or not? What struck him was the dyspnœa. How does an ordinary case of pleurisy begin? It almost always begins with intense pain in the side, worse on drawing breath. Boerhaave, who lived before auscultation and percussion were practised, in his "Aphorisms" says when a man has a pain in the side, worse on drawing breath, he is suffering from pleurisy. That was the way they recognised it before auscultation or percussion were in use. It is worth bearing in mind, even now that we can tell so accurately by means of auscultation and percussion, and it brings out this, that in nearly every case of pleurisy where the pleurisy begins on a particular day, and patients can often tell that, they know it by a severe pain in the side, worse on drawing breath. But that was not the condition in this man at all; he had not a severe pain in the side worse on drawing breath. What he was conscious of was pain in the front of the chest, accompanied by a sense of extreme dyspnœa, very great shortness of breath. Then a patient with a large pleural effusion will very often be in a state of comparative comfort when in bed. I remember very well a woman in Elizabeth Ward who had seven pints of fluid in her left chest. She walked up to Elizabeth Ward—and by some accident she was allowed to walk up instead of being carried up—and when she reached the ward she had marked dyspnœa. She went to bed and was in bed two or three days before tapping. Tapping was not so much a matter of course as it is at the present time. She was in a state of dyspnœa, but though her breathing was affected she did not feel alarmed as she lay in bed. Seven pints of fluid were let out. This man does not show any of that power of becoming accustomed to a large effusion which cases of simple pleural effusion very often do. Before his last tapping I saw him, and he was in a state of great pain as well as dyspnœa. The pain was due to his feeling that he might at any moment be suffocated, not pain in his ribs or intercostal muscles. That, again, is a characteristic of something different from a simple pleurisy. So, taking all those signs together, I believe the man has a growth affecting his left lung and left pleura. Is there any other source of error which I ought to discuss with you? Because I thought it worth while that you should consider in this case while we were still in that state of uncertainty in which you must always be with regard to so obscure a disease while the patient is living, if there is any possible source of error. Could that be a tuberculous condition? I think we may say no. I have never seen as deeply stained and constant an effusion and all the other conditions I have mentioned to you merely from tubercle of the pleura; and you will bear in mind I said that when he does cough something up it does not contain tubercle bacilli.

Could it be another kind of tumour altogether? Could it be aneurysm pressing on the root of the left lung? That is in the highest degree improbable. It must be exercising very considerable pressure and therefore it must be a large aneurysm if it is one at all. But there are no physical signs pointing in that direction. Moreover, if it were an aneurysm it must have been, with all that blood coming away, leaking for at least five weeks. One may therefore be practically certain that it is not an aneurysm.

What can you do for such a patient? Of course all you can do is to try and relieve his symptoms and tap him whenever his dyspnœa is urgent, and feed him. As long as he is not suffering from dyspnœa and as long as the effusion is not very large you can see that he does not suffer very much. He is somewhat wasted, but he is able to take his food, and that is a good illustration of the fact of which it is very important for the comfort of your patients and their friends that you should have plenty of examples in your mind, that new growths, cancers of whatever form, very often go right through their course without causing pain.

There is one other point I want to mention with regard to the case. It illustrates another principle that also applies to new growths, and that is that their duration is never a very long one. If that man had said he had had dyspnœa for six years one would know for certain that he had not a growth. He has probably had that growth for about three months. What will its duration be? It is very rare indeed for a patient with a growth of the lung to live as long as a year. A very large proportion of them do not live more than six months from the time the symptoms begin to develop. I thought it was worth while for you to see this example of an uncommon disease.

December 16th, 1907.

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TWO LECTURES
ON
DYSENTERY.

Delivered at St. Thomas's Hospital.

By F. M. SANDWITH, M.D., F.R.C.P.,
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LECTURE I.

GENTLEMEN,—Before I begin to talk about dysentery I think it will be well to pass round for your inspection a book which shows pictures of the intestine in a very early stage of the disease. The specimens in the bottles which I have on the table will enable you to tell what the lesions look like at a later stage.

It is about a third of a century since I was sitting in this theatre listening to Dr. Murchison's lectures on medicine. I expect you have often heard that he was one of the best teachers, if not quite the best teacher of his day, and from his Indian experience he was almost the only physician in London who was able at that time to lecture from personal knowledge on tropical diseases. He used to tell us that dysentery was a disease which usually began with fever and diarrhœa, and was distinguished from other forms of acute disease of the large intestine by the presence of tenesmus and of stools containing blood and mucus, often consisting of blood and mucus alone, without any fæcal material. To-day there is very little to be added to this description, though one ought to state that dysentery is now regarded as a clinical expression for a group of different diseases.

Varieties.—Some people think that there are very many different forms of the disease, but I think it will be enough for us to-day to divide them into bacillary, amœbic, and pseudo-dysentery. By pseudo-dysentery I mean dysenteric diarrhœa in which neither bacilli nor amœbæ can be found. All the varieties betray some clinical resemblance, though they may differ widely in their cause, duration, morbid anatomy, complications, and geographical distribution. It was not necessary in historical times to go as far as the tropics to meet with dysentery, for as late as the seventeenth century dysentery was endemic in Ireland, though it has always been a disease of rare occurrence in England. In 1682 Boate wrote: "The looseness does also greatly reign in Ireland, as well among those of the country as among the strangers, wherefore the English inhabitants have given it the name of the Country Disease." And Borlase wrote in 1679: "The dysentery or flux is commonly called the country disease, and well it might, for it reigns nowhere so epidemically as it doth in Ireland." Dysentery is to-day a much less terrible disease than it was in former times, and we never hear now of general epidemics spreading over areas as large as half Europe, though there may still be widespread outbreaks succeeding both war and famine. Fifty years ago dysentery and ophthalmia were the two real endemic diseases of Egypt, and in Cairo Griesinger found that more than half of his autopsies showed signs of dysentery. That is to say there were 186 cases in 363 bodies examined; 96 of these 186 actually died from dysentery, while in the remaining 90 death was due to some other disease, of which dysentery was only a secondary complication. Professor Symmers performed 460 autopsies in Cairo in 1901–1902, and found only 18 cases of dysentery. His figures therefore show less than 4 per cent. as compared with about 26 per cent. of Griesinger's half a century ago. This lecture will be chiefly taken up in discussing the bacterial or bacillary form of dysentery, which may occur as an endemic, an epidemic, or a sporadic disease. In 1901 a small outbreak occurred at Aldershot which is supposed to have been caused by importation of the disease from South Africa; there were 170 cases and 38 deaths in a population of 31,000 people. Such outbreaks are still common in many parts of Europe and in tropical and sub-tropical countries. Everywhere there is a risk of groups of adults living in institutions becoming attacked; such as in barracks, camps, lunatic asylums, and prisons. Even in your student days there have been such outbreaks in this country, and Shiga's bacillus is said to have been found in the fæces. Endemic dysentery is now confined to warm climates, and sporadic cases are, of course, those which occasionally occur in places where the disease is usually unknown. Dysentery has been known in every inhabited part of the world from the equator to the poles, but it increases in frequency as one approaches within 40° north or

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south of the equator. Dr. Felkin's map shows you how prevalent the disease is in that area, for it forms roughly a belt or zone around the world, though this does not mean that dysentery is never found in other places. It is some three times more fatal in the south than in the north of France, and it causes seven times more deaths in the south than in the north of Italy. The same relation to latitude is reported from the United States, but on the whole the influence of height above the sea level is more marked on the prevalence of the disease in hot countries than are the changes of latitude. When other things are equal, the dysentery sick rate decreases as the altitude increases.

Three fourths of the outbreaks of dysentery occur in the summer, or in the summer and autumn, though in no part of the year is the disease certain to be absent. Tropical rains have a tendency to increase the disease, because they so often lead to pollution of the soil and drinking water, and the old statement is apparently true, that in the tropics generally dysentery prevails most and is most fatal on moist alluvial soils. The influence of drinking water on the etiology of dysentery cannot be over-estimated. If you have not done so before, I want you to label dysentery as a water-borne disease, like typhoid fever and cholera. The constant association of the disease with campaigns in warm weather is due to want of care in protecting the drinking water, for the most usual cause of dysentery during war is the drinking of surface water which has been polluted by fæces. This was true during the late war in South Africa, and to a more limited extent in the Russo-Japanese campaign. On one occasion during our re-conquest of the Sudan an English regiment, at the end of a forced march in hot weather, near Dongola, exhausted all the drinking water, and, maddened by thirst, the men rushed, in spite of all restraint, to an obviously unclean pond which they came upon towards the end of the day. As a direct consequence of this act some 200 soldiers contracted either dysentery or enteric fever, and 52 of them died. Many more than 200 drank on that occasion, which shows that it is only the susceptible who suffer. I should like you also to notice that this pond seemed to contain a double infection, for some of the soldiers became ill in a few days with dysenteric symptoms, while others waited till their return to Cairo to be admitted to hospital with enteric fever, and a few of the most unfortunate contracted both diseases. I should mention, for the information of those of you who do not know it, that in the east the natives use the banks and the circumferences of ponds as a latrine. The death rate from dysentery in the British navy has been reduced from 12·7 to 1·2 per 1000 since distilled water has taken the place of ordinary water for drinking purposes. Dysentery is not solely water-borne, for, as in the case of typhoid fever, food is liable to become contaminated by the fæces of dysenteric patients, by dust and insects, especially the common fly. I have myself seen dysentery as an attendant on famine, typhus, relapsing fever, and war, and to those we must add scurvy, with which it has often been found associated. It is quite possible that the dysentery of military campaigns and of famine depends partly on the improper and irritating food which often has to be eaten. In South Africa, during the first two years of our war, the dysentery admissions amounted to 24,274, and of this number 975 died. I need not tell you that malaria and dysentery are quite distinct infections, but they are often found to prevail in the same places, and malaria predisposes to, and sometimes aggravates, true dysentery. For instance, Major Buchanan found that the average number of deaths from dysentery in Indian prisons was 3·8 per cent., whereas in a malarious year the deaths rose as high as 9 per cent. I have had some experience of the curious connection between these two diseases, and have seen some members of an English household dangerously ill with malaria, while another member, apparently poisoned at the same time, contracted dysentery without malaria. The only way in which the disease is directly contagious from man to man is when an enema syringe has been used for a dysenteric patient and then employed for another patient without being properly cleaned, or when the poison is introduced by contaminated fingers into the mouth. But indirect contagion is fairly common, especially in crowded houses or in unhygienic institutions. Persons suffering from chronic dysentery are capable of spreading the infection to healthy people by means of their stools, so that dysenteric patients returning home from abroad should be treated with suspicion. Besides the possibility

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of infection by the entry of the bacillus into the mouth and nose, and in very rare instances the anus, there is one case on record of the bacillus coming into contact with the conjunctiva and causing dysentery in twenty-four hours. All ages, from the infant in arms to senility, both sexes and all races, are liable to dysentery, but it is more common in children under five than in adults. There is no method of becoming immune from the disease by long residence in endemic centres, though there are a greater number of victims among Englishmen who have recently arrived than among those of long residence in such countries.

Mortality.—The Indian statistics for soldiers and prisoners give a death rate varying from 3 to 4 per cent., but Dr. Andrew Davidson has pointed out that these figures may include a large number of mild cases which are really pseudo-dysentery. According to Shiga, the case-mortality in Japan varies from 16 to 30 per cent., and in Hong-Kong in 1902 the death rate reached as high as 37 per cent. Our true bacteriological knowledge of this form of the disease dates from 1898, when Shiga studied an epidemic of dysentery in Japan. He found that the dysentery bacillus, which is usually named after him, fulfilled the following requirements: (1) It occurred constantly in patients suffering from the disease; (2) it was a species not normally present in the rectum or fæces; (3) it was capable of producing in animals lesions which were similar to those in man; and (4) it showed in cultures the agglutination reaction with the blood of patients suffering from dysentery. But the Shiga bacillus represents a group of types nearly related to one another, and there is a slight difference between the bacilli obtained from Japan and those from the Philippine Islands, though the morphological and cultural characters are similar in both types. The bacillus is a short rod, about the length of the typhoid bacillus, but somewhat plumper, with rounded ends.

The diagnosis of dysentery is based upon the anatomical lesion and the clinical symptoms, which entirely agree wherever the bacillus has been taken from, but we must allow that the term "dysentery bacillus," like the term "cholera bacillus," includes several types, which show variation in their fermentative and agglutinative qualities. When cultures are mixed with fæces in dry air the bacilli live twelve days, and seventeen days in linen soiled with fæces. In water they survive from five to nine days, and in milk, butter, and cheese about nine days. They are killed in half an hour by one per cent., and immediately by 5 per cent. carbolic acid or by a solution of 1 in 2000 corrosive sublimate. You will at once see that it is just as necessary to disinfect or burn dysenteric fæces as in the case of enteric fever.

The Shiga bacillus is now accepted as the etiological organism of epidemic dysentery in every country, and it is often the cause of endemic disease in the tropics, and of some sporadic cases in temperate climates. It may be found all through the intestines, but more frequently in the lower bowel, associated with *Bacillus coli* and other bacteria. Even when it has been impossible to find it in the fæces during life it may be discovered at the autopsy in scrapings of the intestinal mucous membrane, even though there are no visible lesions in that part of the intestine. It can also be isolated from the mesenteric glands, and occasionally from the liver. Professor Flexner, who has worked so thoroughly at this disease in the Rockefeller Institute, New York, says that gentle scraping of the rectal mucous membrane during life will yield the bacillus, even when it has not been isolated from the fæces. We do not know how the micro-organism lives outside the human body, but it has been found in many children suffering from summer diarrhœa. This is a significant fact. The crucial experiment of producing dysentery in man by the swallowing of cultures of the dysentery bacillus was made by Strong and Musgrave on a Filipino prisoner who had been condemned to death. The immediate result was the appearance of characteristic dysentery, from which recovery took place, and from the mucus in the man's stools the bacillus was obtained in cultures.

I now want to refer to the morbid anatomy. In the acute disease the intestines may show the appearances of catarrh only, of false membrane, of ulceration, and sometimes of all three conditions in different parts. The catarrhal condition is by no means peculiar to dysentery, and will probably be present in both the small and large intestine. It is usually the flexures of the colon, the cæcum, and the rectum which show the most severe and advanced lesions of dysentery, and these spots are believed to be affected chiefly because the fæces

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there remain in contact for a longer time with the mucous membrane. But in virulent cases I have seen the mucous membrane of the whole of the colon involved uniformly. In an ordinary acute case the lesions are first seen on the elevation of the mucous membrane folds, and from here they stretch to lower levels. You will now think of the pictures which I passed round, showing little tiny dots. Every now and then you will see a fresh dot, which means a beginning of the disease and the earliest possible necrosis. Those patients happened to die before the disease reached any further stage. There will probably be a swelling and necrosis of the mucous membrane with the formation of false membrane. The mucous membrane in the neighbourhood of the ulcers or patches of false membrane is congested and œdematous and covered with blood-stained mucus, not at all unlike the white of a raw egg which has become pinkish by blood-stains. The false membrane gradually extends in an irregular shape, and becomes dark and otherwise discoloured, perhaps dark green, as you will see in the pictures. And the solitary lymphatic glands become swollen and rather apt to soften in the centre, forming dozens of minute ulcers. This is in the great intestine, while in the small intestine you sometimes find a little inflammation but usually no ulcers. Ulceration of the mucous membrane occurs because of the separation of the necrotic spots and the false membrane. Consequently the ulcers depend, both with regard to area and depth, upon the degree of necrosis which has already taken place. It must not be thought that the ulcers are confined to the mucous membrane. As you will see in some of these sections and in some of the museum specimens, the submucous tissue has disappeared, and eventually all the coats of the intestine may be destroyed, so that the serous lining is perforated. This perforation is not invariably fatal, because inflammation of the peritoneum may lead to adhesion of neighbouring coils of intestine, shutting off the local suppuration and producing a permanent thickening of the intestine with adhesions. Here is a specimen in which there are two perforations, but the catalogue says it is doubtful whether they were produced artificially or not. It really does not matter, because if you look at the specimen you will see that the parts are extremely thin, like tissue-paper, so that even if the wall were not perforated before death it might easily have been so. No physician can form any impression as to what is probably taking place in the intestine unless he examines the abdomen regularly with care and delicacy, and also the stools. Where it is possible I prefer to see every motion passed by the patient, even if they number thirty or forty per day. Nature may begin the process of repair at any stage. Besides the changes in the great intestine, which, of course, are the most important, you will find at the post-mortem examination, hyperæmia of the peritoneum, hyperæmia of the mesenteric glands, which are enlarged, and hyperæmia with catarrh of the small intestine. In chronic dysentery the whole intestine is thickened and hardened, with strictures alternating with corresponding dilatations of the gut above the strictures. The serous surface may be pigmented, especially in cases complicated by malaria. The mucous surface may also be pigmented, and the scars of old ulcers can be seen in a fairly recent case. The pigment on the mucous surface is probably due to the iron in the blood producing iron sulphide with the sulphuretted hydrogen of the intestine.

Experimental dysentery.—Flexner has not succeeded in conveying the symptoms or lesions of dysentery to animals by feeding experiments. But the dog develops diarrhœa, and after five or six days dies, when the mucous membrane of the gut may show hyperæmia and small hæmorrhages, while from the mucus the dysentery bacillus may be recovered. This reminds one somewhat of human beings who die in a few days, before necrosis and ulcers have had time to appear. Cats are not susceptible to cultures given by the mouth unless they have been previously subjected to croton oil, when diarrhœa persists till death; though bacilli can be found in the fæces, there are no characteristic lesions. Yet cats, as I shall tell you in the next lecture, have been infected by rectal injections of the amœbic variety of dysentery. So much for feeding experiments. On the other hand, rabbits, like men, are peculiarly sensitive to cultures of the Shiga type of bacillus, and if such cultures are injected, either under the skin or into the veins, fatal diarrhœa results. Their intestinal mucous membrane becomes swollen, œdematous and hæmorrhagic, and is covered in places with false membrane, while animals who survive a few days show ulceration of the intestine.

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Symptoms.—The incubation period of true dysentery varies from twenty-four to forty-eight hours. The early symptoms set in suddenly as a mild diarrhœa, with failing appetite. After a day or two the action of the bowels is accompanied by griping pain, chiefly in the descending colon; the motions become more frequent and relieve the griping pain temporarily, though it is apt to return when the patient makes any movement or takes any food. The early motions consist entirely of fæces; they then become mixed with blood-stained mucus, and soon consist of blood and mucus without any fæces at all. The griping pain, which may be all over the abdomen, is now associated with painful straining, so that the sufferer, in spite of his increasing weakness, is never quite so comfortable as when he is attempting to relieve his rectum. Tenesmus and constant irritation of the anus cause the latter to become inflamed and painful, and the slightest pressure on the colon, especially on the descending portion, causes great pain. In mild cases there may be no fever, in ordinary cases the temperature rises to 100° F. or 103° F. for a few days, while in patients who are dangerously ill the fever may be above 104° F. The appetite is bad, the tongue coated, and the patient suffers much from thirst. The urine is decreased in quantity, and its solids are increased. I must not forget to mention the offensive odour of the motions, which permeates the bedroom of the sick man. In all fatal cases the griping and tenesmus almost disappear before death. The motions are passed involuntarily, the tongue becomes dry, and collapse gradually develops with subnormal temperature and clearness of mind alternating with moderate delirium. In gangrenous cases extensive sloughing takes place, and the patients may die as early as the third or fourth day of the illness, though some few recover. Favourable symptoms betokening recovery are: the motions becoming more healthy, more feculent and diminishing in number; more copious urine; and improvement in the tongue, pulse and temperature.

Chronic dysentery is the result of an acute attack, or a relapse of a persistent nature after an acute attack, or a series of relapses. The patient suffers very much from indigestion, flatulence, debility and anæmia, and he is constantly irritated by a fresh attack of his old enemy. Such patients suffer in this country or any other dysentery-free country. Dysenteric diarrhœa is clinically very similar to true dysentery, but there are seldom more than twenty motions in twenty-four hours. If the fæces of a case, which you have labelled "dysenteric diarrhœa," reveal the presence of Shiga's or Flexner's bacillus, you had better change the diagnosis. Arthritis is an interesting sequel of dysentery, the large joints being chiefly affected. The heart is not involved, and the disease eventually resembles rheumatoid arthritis. The few cases of this kind I have seen have not been cured, though they have not died. Pyæmic complications sometimes occur or the liver may become inflamed, as evidenced by pain, tenderness and weight in the right hypochondrium. But the formation of a hepatic abscess, which you all know is associated with dysentery, is rare in bacillary dysentery.

Now as to the diagnosis. First of all you have to make up your mind whether it is real, true dysentery or a dysenteric diarrhœa, which resembles it. It is only experience which will help you there; certainly no microscope will do it. It does not matter to the patient a bit; all he wants is to be saved the constant desire to empty his rectum. One of the chief things to impress upon you again is the importance of examining the fæces in all chronic cases for the eggs of parasites, such as *Ankylostomum duodenale*, or the Bilharzia worm. And here it is convenient to point out that it is not everyone, certainly not every nurse, who can wash a motion properly. Yet it is simple. Put the vessel containing the motion under a tap or under some other supply of water and wait until the solid parts have time to sink to the bottom. Pour off the supernatant liquid and wash again, and so on until you have arrived at the substratum, which has now been cleaned by the various processes of washing. Thus you will find such mucus as the dysentery motion may contain, you may or may not find worms, and you will probably find certain indigestible articles of food. You must look for eggs under a microscope, because in neither of those worm affections do you expect to find worms in the fæces, unless in ankylostomiasis you have given medicine to remove them. In hospitals it is best to place the motion, or the residue, after the washing, into a covered glass dish, which can be handed to the physician to look at.

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In acute cases of dysentery the bacillus may be isolated from the fæces, and after the first week the agglutination test may distinguish between the bacterial and the amœbic variety of dysentery.

Prognosis.—This must depend on the character of the stools, on how far the general condition of the patient is affected by the local disease, and on the previous habits of the patient, in which alcohol must be taken into account. Among the dangerous symptoms are hiccough, delirium, great prostration, copious hæmorrhage, and, of course, perforation of the intestine.

Prevention.—There must be absolute cleanliness of water, or only water which has been boiled should be used. If you cannot boil the water or do not wish to, please make a note that I know only two reliable filters in the world, the Pasteur-Chamberland, and the Berkfeld. Do not use charcoal filters, which I still see sometimes in England. Always suspect surface-water wells.

Then as to food. Do not be content to remember that the disease is water-borne. Much of your food comes into contact with water, directly or indirectly. Regard especially with suspicion uncooked vegetables, including salads, fruit, milk and butter. You aim at preventing the occurrence of either diarrhœa or constipation, and at preserving the general health in every way you can. Avoid exposure to the sun, wet, cold and fatigue. If you are in charge of troops marching through the enemy's country it is difficult to do these things, but they are pious rules to keep in mind. It is very desirable for individuals to wear a flannel belt, day and night, and remember that your women patients are as liable to be attacked as men. Therefore advise both sexes, and children also, to protect their abdomens by flannel belts. The same precautions should be observed about disinfecting fæces, and about the hands of yourself and the nurses as in typhoid fever. The Japanese prevented dysentery to some extent in their war by sending on in advance sanitary officers to test the purity of the water. I am sure that was of tremendous importance. Another thing which the Japanese did and which may have had some influence, though one cannot quite conceive how, was to issue a bottle of creasote pills to all the soldiers, telling them they would not get dysentery if they took a creasote pill three times a day regularly, and this had a very fine moral effect. What was more important was that the Japanese soldier is so constituted that I am told by eye-witnesses that when the man came round to distribute water, the privates would say to him, "Are you certain this water is good? If not we will go without, because we do not wish to be upset, and become ill, and give up the rest of the campaign." And if you have ever seen the British soldier when he is thirsty you will know that he is not careful in that way. So much the worse for the British and so much the better for the Japanese.

Now, lastly, as to treatment. In the first place the patient must be given absolute rest. There is nothing mysterious about dysenteric ulcers: you know that if an ulcer occurs on the leg you rest it, you wash off any sloughs and get rid of anything dirty, and put on a stimulating lotion or application to try to get the ulcer well. That is what you must do with an ulcer of the intestine, so far as it is a local trouble. The patient must stay in bed for all purposes, having a bed-pan and everything else to obviate his moving. Another thing is to put a cotton-wool pad and flannel bandage on his abdomen to keep it warm. It is very comfortable in diarrhœa, and even more so in dysentery. Then use a hot-water bottle if you can get it, or a special aluminium case made to fit the abdomen—a German invention, or there is a patent article in this country, called Instra, which is an imitation of a Japanese apparatus, in which hot charcoal is continually burning. The diet for a dysenteric patient is very similar to that for typhoid. That is to say, begin with putting the patient on milk, and very often you should use peptonised milk if there is nausea or vomiting. You, of course, will not give stimulating broths, first, because they are not much better than hot water, except as stimulants, and, secondly, because they promote diarrhœa, which you do not want to do. Ipecacuanha has been used as a treatment for dysentery for hundreds of years in Peru. It came to Europe about 1653, when Helvetius cured the Dauphin of France with it, and it was introduced into this country in 1660. It has had various ups and downs since. It was found in those days to be much more useful as the treatment for dysentery than the methods previously employed, which were bleeding, blistering and mercury. The usual method to-day is to precede a dose of ipecacuanha with a preliminary dose of sulphate

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of sodium, and then to give twenty to forty grains of ipecacuanha powder in a bolus. To prevent vomiting a mustard leaf is applied to the epigastrium, and twenty or thirty minims of tincture of opium (for adults only) may be given half an hour before the dose of ipecacuanha. Another thing to remember is that no fluid should be taken for at least two hours after the bolus. Ipecacuanha is still used in some parts of India and in other countries, and therefore it is important to know how to administer it. Lately, membroids have been invented in which five to ten grains are enclosed. It is an animal membrane so prepared and sealed that the contents pass the pylorus without being discharged from their capsule, and are not absorbed until they reach the intestine. The firm who made these membroids asked me if I thought it would be a good thing, and I told them ipecacuanha would be a very good drug to try them with. If you take 10 grains of ipecacuanha on a full stomach you are fairly certain to vomit. A few experiments have been made on volunteers, and it is found that vomiting is either delayed or prevented by putting the ipecacuanha into these membroids. If any of you will come forward after the lecture I shall be pleased to help you to be useful to science. Ipecacuanha has made people vomit ever since it was first known; it was introduced into this country, not as an emetic, but for dysentery, and therefore people have tried to give ipecacuanha without emetin, that is to say, a deemetised ipecacuanha, but it is less good and I do not trust it. In taking away the emetin you take away also some of the other effects which you want for the patient. Nobody can tell you how ipecacuanha acts. It has been suggested it acts by causing the bile to flow, and thus killing the bacillus. If that were so it would be better to trust to an old remedy, which is calomel, and that, of course, is a splendid intestinal antiseptic. You may give it in the old-fashioned way, three or four or five grains at a time. I prefer to give it in the American way, which means one sixth of a grain with a little bicarbonate of soda repeated every hour until the bowels have acted. Opium is not a cure for dysentery, though some people find it very useful. It is a remedy which you can use in small doses in conjunction with other drugs. In malarious dysentery you must use quinine, and you will succeed in curing such cases with quinine only, which have been under treatment by other people for weeks or months without being cured. In hæmorrhage, ice to the abdomen or iced enemata may be used. Another drug which is useful in dysentery is sulphur, half to one drachm three times a day. Bismuth, and especially salicylate of bismuth, which is better than the subnitrate, is good. You must give it in large doses, fifteen grains every four hours, and that should be the mimimum dose. You may, if necessary, give one sixth grain of opium with the bismuth. Betanaphthol is another antiseptic which one is very fond of, and that should be given with bismuth. I have left what I consider the best treatment until the last, that is to say, salines: sulphate of magnesia or sulphate of soda, of which the dose is a drachm every hour until the bowels act. You may have to give seven or eight doses of it before they act. Simarubra is a medicine not well known in this country; it is quite a good tonic after dysentery. Then there is the question whether you should or should not use enemata for subacute and chronic cases. If so you must use large quantities—two or three pints—and you must either raise the buttocks of the patient in bed, or, what is better, raise the foot of the bed eighteen inches or two feet in order that the injections may run very slowly high up the bowel, or put the patient in the knee-elbow position with his head low down. Use a tube and funnel—the ordinary Higginson is absolutely useless for this. After the injection wait, if possible, twenty minutes, causing the patient to be as quiet as possible, so as to retain the injection all that time. If there is great pain you can help him very much by giving a grain of cocaine in a suppository half an hour before the enema. There are two things to put into the enemata. A common one is flax or linseed: take one ounce of flax to two pints of warm water, and let it stand a couple of hours before being strained. That is a very comforting enema; it washes out well and it is very pleasant. It is much better than either plain water or salt and water, and more pleasant than soap and water. The curative enema is either nitrate of silver or sulphate of copper in doses of 1 per 1000. Two pints is the minimum quantity.

December 16th, 1907.

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A CLINICAL AFTERNOON AT THE POPLAR HOSPITAL FOR ACCIDENTS.*

Mr. OPENSHAW: This patient is a youth, æt. 18 years, who presents nine inches *shortening of the left leg*. The history is that at the age of three he had some slight injury to that leg, followed by inflammation and suppuration. Since the age of about six he has had many incisions made, and excision of the knee was performed at that age. After those procedures the leg gradually became stiff. Probably at first the ankylosis was fibrous, but gradually became bony, and prior to the ossification of the ankylosing material the knee became acutely flexed as now seen. The leg, he says, has been thus acutely flexed for ten years. I want to point out to the Society that there is nine inches of shortening of this left leg, which is partly accounted for by shortening of the femur, and partly by shortening of the tibia. There still remains a little of the external tuberosity of the tibia, and a portion also of the condyles of the femur. The epiphyses, therefore, have not been wholly removed, and yet there is this excessive shortening. Now, it is my firm belief that it is wise, if possible, to avoid excising either the hip or the knee-joint until a much later period of life. I deprecate these *early* operations on tubercular joints. Here I may say I do not believe in Thomas's knee splint, because it does not keep the joint sufficiently at rest. If these joints are kept at rest in plaster-of-Paris, which I consider is the only satisfactory way of immobilising a knee for a sufficiently long time, these excisions of hip and knee will become less and less frequent. Seventeen years ago, when I first came on as assistant surgeon at the London Hospital, I did an excision of the hip nearly every week, whereas now I rarely have to do one, and that is due to the fact that I now keep these cases at rest for a much longer period than was the custom formerly. In former days it was the customary practice to cut into the hip or knee-joint whenever there was acute inflammation with abscess formation. We first incised, and if the discharge continued we excised. Damage to the epiphyses in every case resulted, and when the patient grew up there was found to be shortening, often excessive. I show this case to illustrate the amount of shortening which may ensue when excision of the knee is performed early in life. When this patient stands with the pelvis horizontal you will see there is fully nine inches of actual shortening, and this is still further increased by spurious shortening due to his tilting up the left hip so as to throw the weight of the body upon the sound leg.

Mr. POLAND asked if Mr. Openshaw had a skiagram of the condition, as it would be interesting to see if there were any remains of the outer tuberosity of the tibia or of the inner condyle of the femur, as suggested. Both the tibia and the femur were much damaged at their epiphysial lines and arrested in growth. He agreed that the original condition was probably tubercular.

Mr. OPENSHAW: I am sorry I have not the skiagraph with me. Another point is that although this knee is at the present time in a condition of bony ankylosis, for many years it was not so firmly fixed. In many such cases a long period of time elapses before the ankylosing material becomes so firm that it is impossible to bend the knee. Though you may, after excision of the knee, keep the leg straight for two years, it may later become flexed and may be found bonily ankylosed when excised five or more years later. Therefore it is necessary to watch a case for at least two years after excision of the knee has been carried out, and in many cases it is wise to remove the hamstring muscles. I can produce nearly fifty cases like this, I am sorry to say, and I could collect 250 records of cases in which shortening was due to disease of one bone only, and in which the shortening is more than five inches.

Mr. POLAND: I agree with Mr. Openshaw that the practice is to less and less frequently excise tuberculous joints. When I was a younger enthusiast, years ago, it was the custom to excise all these joints both hip and knee, but now we do not. We find in a large proportion of them that if we put on a mechanical apparatus and allow the patient to walk about in the fresh air, a vast number of these cases, even amongst the poor in the East End of London, get well. That, I think, is the chief lesson which Mr. Openshaw intends to bring out by showing these cases. Professor Marsh wrote

* A meeting of the Hunterian Society, with Mr. JOHN POLAND, President, in the chair.

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a paper a little time ago on that point, in which he says the same thing. I agree with Mr. Openshaw's remarks about the old days of surgery, when in the case of a suppurating joint the question which arose was whether there should be amputation or not. We now know that we must not excise a joint simply because it is tuberculous. I have given the practice up entirely, and I do not suppose I have done one case of the kind for many years. I perform the operation now only when there is a disease of the bone, about the lower end of the femur or tibia with suppuration, and then the surgeon clears out the whole joint and diseased bone. It is a question of excision and complete removal of all diseased bone and tissues *versus* amputation.

Mr. OPENSHAW: This is a girl with *double congenital dislocation of the hip*, cured by Lorenz's method. So far as it is attainable I think this girl's case may be regarded as an anatomical reposition of both hips by Lorenz's method. Not only is the function perfect, but the relation of the great trochanter in regard to the anterior superior spine is normal. The lordosis which was present has practically gone, the flattening of the buttock is less, and there is no difference in the length of the two legs. The relation of the great trochanter to Nelaton's line is normal. You will agree, I think, that she walks perfectly. It is two years since I commenced to treat the case. For six months her legs were kept fully abducted; during the following six months the legs were gradually brought down into the normal position, and for a further period of six months they remained in plaster—eighteen months in all. Any patient under the age of six suffering from congenital dislocation of the hip can almost certainly be cured. In regard to the kind of result obtained my experience is approximately as follows: Of 20 cases, I should think that in 6 both functionally and anatomically the result is a perfect one, in 8 or 9 the result is functionally perfect but not anatomically—that is to say, the head of the bone will rest a little external or anterior to the level of the acetabulum, but movement and gait are practically perfect. In the remaining 5 out of the 20 you will get re-dislocations, either anterior or posterior, or ankylosis with the limb abducted, adducted, or rotated out with more or less firm fibrous ankylosis of the joint. Taken altogether there is no question whatever that these hips, if you catch the patient under the age of six, can be replaced by Lorenz's method. The ultimate result in a given case depends more upon the amount of rotation which has already taken place of the head and neck upon the upper end of the femur than upon anything else. In a double dislocation of the hip there is always rotation of the head and neck of the femur upon the top of the shaft of the femur, on one side or other, sometimes both, sometimes more on one side than on the other. Always on one side there is rotation of the head of the bone, and it depends on that amount of rotation whether you can replace and keep in position the head of the bone, or not. You must kindly take my word for it that that joint was dislocated; I am sorry I have not brought the skiagraph of it. There was one to one and a half inches of shortening. It was kept for eighteen months in plaster-of-Paris. Lorenz, when last in England, taught us that the leg should be kept abducted for six months. I have kept all my cases since then thus fully abducted, but I have come to the conclusion that six months is too long a period. My own belief is that four months is safer. Full abduction for six months produces a rotation outwards of the femur, which is very difficult to correct afterwards. In fact I have been obliged to devise an appliance in order to remedy the rotation outwards of the limb thus produced.

Mr. POLAND: This is a most interesting case, because Mr. Openshaw says it is perfect functionally on both sides. We have seen the child walk, so we know that it is so. But, as he says, such physiologically perfect results are not always anatomically perfect. I would call the attention of those present to the excellent result on the right side; you can feel the head of the femur in the acetabulum in front, whereas on the left side it has not come quite forward. So anatomically on the left side the joint is not perfect. In these cases you cannot expect a perfect joint to be formed every time; if you have a perfect joint on one side it often happens that you have not on the other.

Mr. OPENSHAW: In a bilateral dislocation there may be no rotation of the shaft of the femur on the one side, but on the other side the head and neck of the bone will be rotated, so that the

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articular surface of the head will be directed forwards instead of inwards. Consequently the legs must be isolated inwards, and the toes made to point inwards if the articular surface of the head is to rest against the acetabulum. Thus one leg is in its proper axis with regard to the body, while the other is turned inwards when the head of the bone is in, or rather on, the acetabulum. It is not possible to replace the head of the femur *into* the acetabulum. I have opened quite a dozen of these joints, and it is with the greatest difficulty one can get even the tip of one's forefinger through the constricted capsule which covers the acetabulum.

I now show you another case, a boy, æt. 13 years, with *ankylosis* which is said to be rheumatic in origin. Many forms of arthritis come on with acute fever. The acute febrile condition may be called rheumatism, but I think it often is due to sepsis. I doubt the diagnosis of rheumatism in this case. When the boy first came under my care, the knees, ankles and hips were firmly ankylosed; there was no movement whatever in either of them. The legs were rotated outwards at the hip-joints with the knees wide apart, and slightly flexed. He had not walked for five years, and for most of the time had been absolutely bed-ridden, With the object of correcting the external rotation of the legs at the hip-joints I put the legs into plaster-of-Paris from the toes to the thighs, and incorporated with the plaster a band of steel which rested against the back of each leg and sole of the foot and projected beyond the toes. The projecting ends of these steel bands I fastened together by means of elastic bands, and thus very slowly (in four months) the legs were rotated inwards, the eversion being corrected and the patellæ looking forwards. The hips and the knees were next frequently manipulated under an anæsthetic. I next had each leg fitted with this elastic traction appliance. Each appliance is jointed at the knee and is so arranged that the elastic band tends to extend the knee. The tendency of the knee is to become flexed in walking owing to the body weight. The elastic traction and the body weight acting alternately therefore tend to maintain and increase the mobility of the knee-joint. The mobility was accelerated by occasional passive motion under an anæsthetic. This patient has gradually increased the extent of the movements in all his joints. The movement did not adversely affect any joint except the left knee-joint, which became furiously inflamed and suppurated, and I began to fear that excision or amputation would become necessary. Under Scott's ointment and fixation, however, the suppuration decreased and the sinus healed. On the subsidence of the acutest phase of the inflammation movement of the appliance at the knee-joint was again permitted, and the range of movement has since steadily increased in this left knee-joint, as well as in every other joint. It is only by means of an apparatus such as this which fits the leg completely that it is possible to apply elastic traction to the front of the knee-joint without unduly pressing on the back of the thigh or the calf. When there is accurate fitting you can apply an enormous amount of force to straighten the leg. I have a patient with double osteo-arthritis of the knees, which were each flexed and fibrously ankylosed at a right angle. For four years he had not walked, he had to be carried into the city, and it has taken me a year to straighten his legs sufficiently to enable him to stand upright. He walked round the room for the first time about a month ago. His legs have been straightened by means of a similar splint case appliance.

I now show you a girl with *congenital absence of the fibula*. The skiagram shows that only an inch of the lower end of the fibula is present. This patient's gait is perfect. Near the centre of the anterior border of the tibia there is present a linear depression. I have very rarely seen a case of congenital absence of the fibula which has not exhibited this scar on the front of the tibia.

Mr. POLAND: I agree that there is always a scar or dimple over the tibia where the skin is bound down. I have seen it in the forearm, too, where there has been absence of bone and dimpling of the skin over cicatrices which were bound down to the bone. This condition has been almost invariable in my experience. It is a question of intra-uterine pressure.

Mr. OPENSHAW: There is one and three quarter inches of shortening of the left leg. My own view is that the scar is definitely due to intra-uterine fracture.

Mr. POLAND: I hope Mr. Openshaw will bring this case forward at a future meeting of the Hunterian Society, perhaps with others bearing on the same point by other Fellows of the Society.

Mr. OPENSHAW: This patient exhibits an extremely rare deformity, viz. *congenital shortening of the femur*. This X-ray photograph shows the

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shaft of the femur to be shortened, the condyles and the head being normal in size and outline as compared with the opposite leg. The fibula is also congenitally absent in this case.

Mr. POLAND: These cases of arrest of growth in the femur are curious cases, and I do not understand their ætiology. I have had many of them under observation, and I have tried to ascertain about intra-uterine fracture or any other accident which may have occurred but have never obtained a history of anything of the sort. Here the condition is complicated by absence of the fibula. I would like to hear from Mr. Openshaw whether he assigns any cause for the enormous arrest of growth. The femur is absurdly small.

Mr. OPENSHAW: I have no opinion to offer on that point, except that it is curious there is no dimpling in the tibia in this case. This is, I think, the only case I have seen of congenital absence of fibula where there has been no dimpling. It may be that the injury has fallen upon the femur and the fibula has escaped. I now show you the case of a girl, of about sixteen years of age, with rigidity of the cervical spine, with a good deal of left, convex, cervical scoliosis. The head is rotated to the right and her neck is quite rigid, the cervical vertebræ being apparently quite immovable. The case is one of acute spondylitis, possibly of rheumatic causation, and of two months' duration. I have really brought this case for diagnosis, and I should also like to hear suggestions as to treatment. The rigidity is osseous in origin and is not due to muscular spasm.

Mr. POLAND: She seems to be able to bend the head a little, and it is mostly the rotation which is difficult of accomplishment. I think it is the articular processes which are at fault, and that is what you find in rheumatic affections of the spine. The articular surfaces are affected more than any other part.

Dr. FORTESCUE FOX: I think it looks like a rheumatic ankylosis. It came on gradually and is said to have followed exposure to cold and draught.

Mr. POLAND: One has often seen the whole spine affected with ankylosis in the same way in adults without any other disease whatever, and there has been no other part of the body involved.

Mr. OPENSHAW: I am now able to show you a child, æt. 4 months, who presents *congenital absence of both tibiæ*, complete absence of the left and the lower half of the right. The upper portion of the right tibia ends in a sharp conical point. Immediately below this sharp conical point there is another portion of the tibia. The conical point corresponds exactly with a dimple on the front of the leg. The tibia has been fractured in the middle. This case would seem definitely to explain the presence of the dimple in the centre of the leg in these cases of congenital absence of one bone.

Mr. POLAND: I think this case confirms the view which Mr. Openshaw has expressed as to intra-uterine fracture.

Mr. HUGH RIGBY: I have brought this boy, æt. 17 years, chiefly in order that I may receive some suggestions as to treatment, and also because he presents features of interest. He complains that he lost his work because he could not turn his forearms. One finds that they are almost completely ankylosed in the pronated position. In the right arm there is practically no power of supination at all. On examining the upper part of the forearm I find the head of the radius is absent from its normal position and is displaced forwards in front of the external condyle. There is some limitation of the power of flexion. In the left arm one cannot discern any head of the radius, and there is nothing in front corresponding to it to be felt. Apparently he has complete fixation in the pronated position. I want the opinion of Fellows as to whether I can do anything to restore the powers of pronation and supination.

Mr. POLAND: The head of the radius is small. Do you consider the causation is rheumatism?

Mr. RIGBY: No, I think it is a congenital condition. In the radiograph it looks as if the upper ends of the radius and ulna were fused. Even if we did any operation we should be handicapped, I take it, by the muscles being so much atrophied.

Mr. OPENSHAW: I have seen dislocation of the head of the radius produced as a result of paralysis. But this case does not seem to be of that nature. It seems to me to have been produced by arthritis of the radio-humeral articulation, leading to disintegration of the ligaments and ultimately to dislocation. There is absolute ankylosis of the upper radio-ulnar articulation, as well as dislocation of the head of the radius forwards. I think the ankylosis came secondarily to the dislocation. I think there was arthritis, erosion of ligaments, dislocation, and then ankylosis, in that order, the ankylosis taking place with the forearm pronated. On the left side there has been absorption of the head of the radius and of the epiphysis. I should not hesitate to try and remedy this, carefully avoiding the main nerves. I think the atrophy of muscles must be mainly due to disuse, and I do not see any evidence of paralysis whatever. He moves his fingers well, there is no wrist drop, and no excessive wasting of any group of muscles. I think it is arthritic ankylosis of the joints, and that it should be operated upon. In the paralytic cases I have cut down on the head of the pronator radii teres and detached the insertion of that muscle from the radius, and passed it through between the radius and ulna, and in that way made it into a supinator muscle, with marked benefit.

(*To be concluded.*)

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** Specially reported for the Clinical Journal. ' Revised by the Author.*

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THE CLINICAL EVIDENCE REQUISITE BEFORE AND AFTER THE PERFORMANCE OF SURGICAL OPERATIONS.

A Clinical Lecture delivered at St. Bartholomew's Hospital.

By C. B. LOCKWOOD, F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—Everyone in this theatre will be called upon to determine whether operations ought to be performed or not. Perhaps he will be called upon to perform them himself. And further, he will have to take charge of cases after operations have been performed. Therefore it is clearly requisite that you should all have clear notions in your minds how to form a proper estimate of the condition of the patients before the operation, and you ought also to know how things are proceeding afterwards. I have observed as regards these points that the gentlemen who attend my classes are not always very clear in their minds. Indeed, I over and over again find that, when shown a temperature chart, some are unable to read it; that is to say they are not able to tell what the markings on it mean.

For our purposes we may classify operations into, first, those of immediate necessity. With these I am not at present concerned. If a person is choking or suffocating we may have to perform an operation instantly, quite irrespective of the condition of the heart, kidneys, and so forth. Then there are others which are operations of ordinary necessity. For instance, when a carcinoma of the breast has to be removed. But here the time element is not pressing, and we can proceed at leisure on definite lines, and form a preliminary estimate of the condition of the patient. And lastly, there are operations which I shall call operations of expediency, which are not done to

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save life, nor to prolong life, but to make life more bearable, more agreeable, and perhaps to fit the patient to enter one of the public services. An ordinary instance would be an operation for varicocele, or an operation for the radical cure of hernia, and perhaps also some of the operations on the joints. These last are a class of case in which above all it is essential that you should form a thorough and competent estimate of the general condition of the patient, and be certain that you are not likely, after the operation has been performed, to be confronted with some crisis owing to your having overlooked disease of an important organ. In forming your estimate of the general condition of the patient you will be guided, not by subtleties, or by things which are difficult to ascertain, but by broad, simple, obvious features. You know well our method of investigation. And the first step is to look at the whole patient. And you will acquire—perhaps you have already acquired—a habit of forming a rapid estimate of the condition of the patient from what you see at first glance. Wasting of the muscles of the neck would be significant, and cause you to make a careful investigation of the whole of that patient's body. Anæmia would place you on your guard, and other things which are seen at a glance when first the patient is before you. Other things, too, have to be considered. The age of the patient is put down in the notes, but the significance of that is not always appreciated. You will have to make up your minds what weight age ought to carry. A patient will often say, "I am too old for an operation," and it will be necessary for you to have thought out the effects of age. I do not think that the age in these aseptic days is of so much consequence provided that the aged patient has sound and healthy organs. And it is remarkable what severe and prolonged operations those of considerable age will pass through provided their organs are sound and healthy. The occupation will often, when ascertained, throw considerable light upon the dangers. For instance, the class of people who drive cars usually drink quantities of alcohol, and seldom have sound hearts, kidneys, and arteries. If I were asked who were the most dangerous members of our community I should specify people who drive mineral-water vans, or "minerals," as they call them, because these people go round to the various public-houses and the publicans, from mistaken motives of kindness, provide them with ardent spirits to drink. Incidentally, in examining the whole body of the patient you will probably make a remark upon the condition of the pulse. What you find might put you on your guard as regards the heart and arteries. A rapid pulse which was not simply due to the excitement of the moment ought to make you cautious. It may be due to Graves' disease, or one of the infective diseases, so that you take the temperature, and possibly proceed to investigate the condition of the blood itself. The rate of the respirations is not sufficiently taken note of. In consequence of the rapidity of respiration not having been noted pneumonia has been overlooked, and the abdomen has been opened unnecessarily. At the beginning of pneumonia the abdominal wall may be painful and rigid, so the abdomen is supposed to be the seat of peritonitis. Assuming that you had learnt to form a proper estimate of the general condition of the patient, you now enter into more minute particulars, and will now proceed, as in all of our investigations, in proper anatomical order. I suppose you would therefore begin at the surface of the body, and that is not infrequently of some importance in regard to the performance of an operation. If the skin is rough and in bad condition it will be more difficult to disinfect. Recently a child was discharged from Kenton Ward which required an operation of expediency performed, and it was refused because of persistent impetigo. Sometimes ulcers are present, and, although I may not be right in my opinion, if there is a septic ulcer—for instance, associated with carcinoma of the breast—I do not believe any human being can disinfect that ulcer, and therefore it follows that I do not believe that any human being can do an aseptic operation. In other words, when an ulcer is present and an operation of necessity has to be performed, it is clear that the dangers of that operation will be vastly increased. I am speaking from memory, but the only cases which I have lost after operations for cancer of the breast had septic ulcers at the time of the operation. I need hardly say that, supposing on the surface of the body there is a leak from one of the septic canals—from the alimentary canal for instance—there is again grave danger from the operation, especially if you try to close the wound which you had made in the vicinity. Assuming that we have glanced at the

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surface of the body, you will have now to proceed to form an estimate of the general condition of the patient from other particulars. And my memory carries me back to the time when Sir William Savory used to call our attention to that which he called the tripod of life. He said if the tripod of life was sound, operations could be performed with safety, that tripod being the heart, lungs, and kidneys. If you do not, from habit, examine the heart of the patient before operation, you are sure at some time or another to meet with disaster, or at all events have serious alarms during and after the operation. Perhaps the worst form of cardiac disease is disease of the aortic valves, and I can remember many years ago having to amputate through the thigh of a man who had very serious aortic disease. When the bone was sawn through the man gave a gasp, his heart ceased to beat, he ceased to bleed, and we thought he was dead. But, fortunately, the heart went on again. In that instance disease of the aortic valves was known. But I remember once operating for an irreducible hernia. The man was rather elderly, and the operation had to be done because he could not wear a truss, and therefore was in danger of strangulation. Also the rupture interfered with his occupation. I proceeded to do the operation, and it had no unusual features. But afterwards he became breathless, and his pulse became intermittent, and nearly stopped, and then I discovered what I ought to have known before, that he had a systolic murmur. Had that been known before I should still have performed the operation, and perhaps should not have taken any additional precautions, but you will agree with me that I ought to have known. In examining the heart you sometimes find that the patient has an intermittent pulse. I do not think an intermittent pulse has any bearing on the question of operation provided it is not associated with any actual disease of the heart. I have several times observed children of ten or twelve years of age to have intermittent pulses. And of course the question arises whether that increases the danger of the operation. My friend, Dr. Alexander Morison, settled that question for me once in an interesting manner, because he pointed out that in the case of a small boy who had a very intermittent pulse when he was lying down, the beat of the pulse became regular and steady when he stood up and walked about the room. In other words, this boy's heart intermitted when it had not much work to do, but when it had adequate work to perform it steadied itself and became regular and equal to its task. I will not refer in any detail to the condition of the arteries. If, on feeling a person's pulse, you ascertain that the arteries are hard or rigid, obviously it has a bearing on your views concerning operation. It ought to lead you at once to make a strict and careful examination of the condition of the kidneys. Where the arteries are calcareous I think you would have to be very cautious how you perform any long and dangerous operation. Calcareous arteries, of course, are more difficult to tie and more apt to bleed than normal arteries. Besides, the coronary artery may be calcareous as well as the others. I have already referred to the importance of the respiration, and here I would say it is rather a serious thing (taking the next item in the tripod of life, the lungs) if you overlook the fact that the patient is suffering from bronchitis. There is no question at all that bronchitis would be very much aggravated by prolonged anæsthesia, especially if the anæsthesia is conducted by one addicted to giving ether. Ether is an irritant. We object to ether as an anæsthetic, both because the patients dislike it so, and because it is so apt to irritate their lungs. The operation of expediency may be for a hernia or for the removal of a chronically inflamed appendix. And if you operate on a patient who has bronchitis, his sufferings are very much increased on account of the constant disturbance of the wound by the coughing, and that is not very conducive to a successful result. Wounds which are continually strained by coughing are very apt to stretch and give way. I have already referred to the importance of examining the lungs, whether you think the abdominal pain was due to pneumonia or not. During the last two months we have had a patient in Harley Ward who came in with what was supposed to be an attack of peritonitis. The abdomen was exceedingly painful, it was rather distended, and it was tender to the touch. He had a rapid pulse and a very high temperature. However, we observed that his bowels acted with great ease when an enema was given, and flatus was freely passed. We determined to wait and see what occurred.

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In two or three days' time it was clear that he had pneumonia over the base of one of his lungs. It is curious, but the abdominal symptoms often precede the manifestations in the chest.

I now come to the other stem of the tripod of life, and that is the genito-urinary tract, including, of course, the kidneys. I have said already that the points which you are apt to overlook are usually very large and important. When you come to the genito-urinary tract one of the first things to ascertain is how the patient micturates. You would say, "How simple; surely it is hardly necessary to inquire into that." But I do not know whether you have thought that when a man is afflicted with stricture of the urethra, in consequence of his straining he often develops rupture or hæmorrhoids. I have known a case in which a patient was operated upon for hæmorrhoids, and then, as is so common after that operation, had at attack of retention of urine, and when the surgeon came to draw the water off he found that the patient had a tight stricture of the urethra, and that he could not get an instrument into his bladder. Within my own experience I can recall the case of an old gentleman who had hæmorrhoids. I knew perfectly well that he had an enlargement of his prostate, and I objected to perform any operation on the hæmorrhoids because of my fear of genito-urinary trouble. But there were other urgent reasons why the hæmorrhoids should be operated upon, so that, proceeding with the greatest caution, and pointing out the risks to the patient and his friends, the operation was performed. Afterwards an attack of retention of urine occurred, partly owing to the operation and partly owing to the enlarged prostate, but it was got over by the passage of catheters. He began to pass his water himself on the fourth or fifth day, and then the bowels acted. He ceased to pass his water, and catheters had to be passed again. And then he declined to have any more catheters passed, and to save his life a suprapubic opening had to be made. I cannot tell you what a horrible anxiety that case was to me, although I went into it with my eyes wide open. Fortunately he got better, and appears to be perfectly right now. But I would rather not have to undertake such a case again. I said stricture of the urethra sometimes was associated with rupture, on account of the straining. Some years ago a man was operated upon twice for the radical cure of inguinal hernia. On inquiry it was found that he had a very tight stricture of the urethra, and as long as he had that he would, I suppose, go on getting ruptures, and perhaps having radical cures done until he died of something else. Surely a proper and systematic inquiry into that man's micturition would have told that he had a stricture of the urethra. When you come to the urine, I need hardly say that it is necessary to make both a quantitative and qualitative examination. If I wanted to ask any gentlemen in charge of a genito-urinary case a question which he would never answer, I should ask how much urine was being passed in the twenty-four hours? And it is singular that he should so seldom ascertain a fact of the greatest importance. The usual account is read out: urine is acid, specific gravity 1020, and so on. Then comes the question; well, how much has been passed in twenty-four hours? If the specific gravity is 1020 and the patient is passing 20 oz. in the twenty-four hours, there is something to be thought about. That is so with a patient in Lucas Ward, by whom only 20 oz. of urine has been passed in twenty-four hours. We are proceeding with her case, and we have got this far. Assuming that only 20 oz. of urine had been passed in the twenty-four hours, evidently something strange is going on in the kidneys. This patient is supposed to have a stone in the *left* kidney, but no shadow can be seen in the skiagram. We have made an endoscopic examination, and seen a clot coming out of the *right* ureter, so clearly our suspicions formed upon the small quantity of urine are not unwarranted. With regard to the quality of the urine, you should have thought out what bearing any alteration in the quality of the urine will have upon your operative procedure. Perhaps one of the most ordinary alterations in the urine is an addition to its solids, some albumen added to it, showing nephritis. One has frequently had to operate upon patients who have had some degree of chronic nephritis, and they have done so uniformly well that one hardly looks upon that as one of the things to excite apprehension. Obviously the presence of conditions denoting acute nephritis would be a matter of very great importance. Sometimes sugar is unexpectedly found in the urine, and its presence is of grave import. I will put an ordinary case before

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you. A man, æt. 70 years, requires a rather severe operation performed. I need not specify the operation. He comes up from the country, and as part of the routine examination of his case the urine is examined. It is then found to contain 3 per cent. of sugar. And the patient obviously is suffering from diabetes. How are you to proceed in a case of this sort? The patient's life, owing to his condition, is a burden to him, and clearly an operation must be performed if it can be done with reasonable safety. Indeed, it was incumbent upon him to take risks, because the disease from which he was suffering was one which was likely to kill him in any case. A great many questions arise. For instance, would the diabetes make the anæsthetic more dangerous? We went into that carefully and it was agreed that it added slightly to the risk, for the anæsthesia which produced profound sleep might end in diabetic coma. Still that is a rather far-off chance. Next, was there any risk of the operation being followed by diabetic coma or by acetonæmia? We determined to place the patient on a diet without sugar and starches, and see whether the sugar was controlled. If it was easily controlled then one could proceed to perform the operation. That patient recovered from the operation, and in ten days' time was walking about the room, taking an ordinary diet, and making arrangements to go back to the country. But one day I was told that he was drowsy. Next day he was rather more drowsy, and after a time it was clear that he had some of the complications which are associated with diabetes, probably acetonæmia, and he at last became comatose and died. I did not give this patient large doses of carbonate of soda before I performed the operation; perhaps it might not have been politic under the circumstances to have done so, but I think if you knew that a patient was suffering from diabetes you ought to consider whether preliminary treatment by carbonate of soda is desirable. There is one other danger which diabetes entails, and that is the danger of wound infection. I believe it is a fact that a patient with diabetes has sugar throughout his body and in all his tissues, and further, that tissues which are saturated with sugar make better culture media for the various bacteria which may get into wounds. Djabetic gangrene, which attacks the lower extremities, probably occurs because the tissues are altered and laden with sugar and form a better breeding-place for the bacteria, which of course abound in that region, their point of entrance being a trivial wound, such as by cutting a corn or by a scratch.

After the tripod of life, I need hardly say you should pass in review the other great organs. I have not time to go *seriatim* over the abdominal organs, but clearly the condition of the liver is important; so is the spleen, the pancreas, and the alimentary tract. But what I wish to convey to your minds is that if you have thought out your system of examination you can rely upon your general knowledge for suggesting how those organs ought to be investigated. The point which I wish to impress upon you is this: that if you have not thought out a system for yourselves you will overlook some most important facts. I can remember a patient who had enlarged sarcomatous glands removed from the neck. After that, enlargement of the tonsil took place, and the question arose whether that tonsil ought to be removed, the glands having been removed in a satisfactory manner, and the enlargement of the tonsil occurring two years after removal of glands. I thought the tonsil might be removed. But Sir Thomas Smith saw the patient with me, and, wiser and more experienced, he went over the whole of the patient and pointed out growths in the liver which absolutely precluded any operation on the tonsil. It would have been rather tragic if one had removed that tonsil, having forgotten to examine the abdomen, and overlooked the secondary growths in the liver. I have spoken of the abdominal organs, and am now coming to a part of the body which is often overlooked. Your examination is not concluded until you have glanced through the central and peripheral nervous system. The abdominal crises of locomotor ataxy are often mistaken for other abdominal conditions, but ought not to be mistaken if the surgeon was in the habit of passing through his mind the condition of the central nervous system. We had in our wards a patient who was supposed to be suffering from chronic pancreatitis. He was examined by numerous people. The fæces reacted to all the proper tests. After a while his pain in the abdomen diminished. I was very suspicious about him. His nervous system had been examined on more than one occasion, and I asked especially that he should be kept under observation. Two months

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later the house physician reported that he had locomotor ataxy. I believe that the abdomen has been opened for an abdominal crisis due to locomotor ataxy. It is easy to understand how that may happen if you are not in the habit of passing the central nervous system under review. As regards the peripheral nervous system, if you are not in the habit of thinking over the condition of the various nerves in the region of the operation you will occasionally be blamed for things for which you are not responsible. A man had a parotid tumour which had been partly removed elsewhere, and then came to have a further operation for that which remained behind. The question arose as to the condition of the nerves in the vicinity. The facial nerve was paralysed, also he could not hear on that side. Had we performed the operation and not been cognisant of those facts we might reasonably have been blamed for dividing the facial nerve and also for making him deaf. I remember a child who had tuberculous glands removed from his neck. He had had two operations performed. The glands enlarged again and had to be removed. On examination of the various nerves in the vicinity of the incision it was seen that the spinal accessory nerve had been divided, so that there was very marked wasting of the trapezius muscle, and so marked that only a careless person could have overlooked it. Are the parents or the friends sufficiently acute to notice things of that sort? Yes, they are; but he was going in for the army medical examination, and those medical men were sure to observe it, and if you had performed that further operation, and had not noticed that important fact, you might reasonably be blamed for dividing the spinal accessory nerve.

We will assume that the operation has been performed, and I now ask you to give attention to the ordinary sequence of events after it. Some medical men are very much afraid of having operation cases under their charge. I cannot help thinking it is because they do not know what to expect, and next they do not know how to recognise the signs of safety, or the danger signals when they appear. The inevitable sequences of all operations are shock, reaction, traumatic fever, and then recovery or convalescence. Go to the wards and see the patients after operations and see what is done with them. And first of all learn to recognise shock when you see it, and next learn how to estimate the amount of shock which is present in any given patient. You ought also to know what to do in cases of shock, and be perfectly clear in your minds how you intend to proceed, because the procedure is very much the same under all circumstances. Now, reaction. There are a few charts here of those who have had severe operations performed, and you will observe one which I have chosen on purpose, because it indicates what you might expect. A man came in to have gastro-enterostomy performed after copious hæmorrhage from an ulcer of the stomach or duodenum. He was aged, wasted, anæmic, exceedingly ill, feeble, and constantly vomiting. And therefore you might have anticipated he would suffer considerably from shock, especially after a grave operation. So I am not surprised to find that during shock his temperature fell to 97° F. When you proceed to the wards and investigate these people who have shock see whether the pulse is accelerated during shock, or whether it becomes slower. I see the pulse of this patient is put down as 60, but if I were asked I should say that the pulse usually became accelerated during shock, perhaps to 112 or even 120, and so I see it in some of these charts which I have brought down. I think you may say, much shock, much reaction. Here the temperature ran up the same evening to 100·6° F. The temperature of reaction is not always at its height on the same evening, but it may be high the next morning. I have known this occur. The only son of a wealthy father had an operation performed for removal of an inflamed appendix with a concretion in it. The child bore the operation fairly well. The parents were very anxious. Next morning the surgeon is seized upon by the father who says: "Are you aware that my son's temperature is 101° F.?" The answer to that is: "Yes, I am aware of that, and if it was not 101° F. I should feel anxious because he would have had no reaction." After reaction the temperature remains up for a while and that slight elevation of temperature constitutes traumatic fever, and usually during the period of traumatic fever the pulse remains high too. Here is the chart of a case which had rather marked traumatic fever. A short time ago a man's patella was fractured and he had the knee-joint opened and the blood-clot extracted and the dorsal aponeurosis cut away from

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between the fragments, and then the portions of the patella wired together. At the time of the operation we observed that the knee was full of blood, the synovial membrane was much inflamed, and we opined that he would have a sharp attack of traumatic fever. So I am not surprised to see that during the shock his temperature fell to 97° F., that during the reaction it rose to 99° F., and that in the next twenty-eight hours it had gone up to 101° F. Then after moving up and down a little it descended to 97° F. I wonder if those gentlemen who are here would feel happy if they knew that their patient on whom they had performed wiring of a fractured patella had a temperature of 101° F.? But we had expected it because we had seen the blood in the tissues and the inflamed synovial membrane. Also that the temperature of 101° F. was associated with a pulse of 96. This is my crude way of thinking about these things: After an operation, say, of wiring a fractured patella, I see there is a temperature of 101° F., but I do not trouble my head about that until I see what the pulse is, and when I am informed that the pulse is 96 I have no fear. If after that the temperature had gone up to 101° F. and there had been a corresponding acceleration of the pulse, then I should have felt anxious. For instance, here is a disaster. A patient has nephrectomy performed in this hospital. She recovers from that. Afterwards a small blood-cyst or tumour forms in the iliac fossa. That is opened and leaves a small sinus. She then, still with the sinus, goes to a convalescent home, and in a little while she comes back suffering from streptococcic septicæmia. She had been brought into contact with a case of so-called puerperal fever, got from it streptococcic infection, and died of it. About the time she died another patient in this hospital who had an open wound had his pulse quicken. And his temperature went up to 103° F. and the pulse to 112, and we thought this must indicate something ominous, for he had not only a high temperature but also a rapid pulse. The pulse went up to 124 and that was still more ominous: then it went to 136—still worse. That patient also had streptococcic infection and died. So observe that after an operation there is a great deal for you gentlemen to consider and to learn about. If you do not know that the inflammation caused by an operation and presence of blood in the tissues will send the temperature up, at the same time accelerating the pulse, you will be sure to have some anxious moments. I cannot proceed to detail all the sequelæ of an operation, but if it is an abdominal operation which has been performed there are two things I would ask you to pay great attention to, one is vomiting and the other is flatulent distension. After all operations, vomiting is usually a distressing feature, and if the vomiting persists it becomes not only distressing but also dangerous. And it will be your duty to search the patient to see if you can find some cause for it. After abdominal operations one of the most common causes of continuance of vomiting is flatulent distension, which should not be allowed to occur. If it does occur you should be sufficiently skilful to overcome it. Vomiting after the anæsthetic we get over easily by giving large quantities of hot water with soda in it, and allowing the patient to vomit it up again. But I am referring particularly to the vomiting which persists. If it does persist you will have to find some cause for the flatulent distension. That is seldom allowed to occur now, and we prevent it by preparing patients with the greatest possible care for the operation, and we begin within four hours of the operation to prevent the accumulation of intestinal gases. Obviously there are many things to consider after operations besides those I have mentioned. No one will forget after an operation to inquire whether the urine has been passed, and I would only warn you that if urine is retained there are few more critical operations in surgery than the passage of a catheter. I do not know anything more dangerous or anything more tedious than genito-urinary sepsis.

December 23rd, 1907.

Tuberculin as a Diagnostic Measure in Gynæcology.—Pankow's experience with thirty-two cases in which tuberculin was used to determine the presence of tuberculosis was not much in favour of the tuberculin test in gynæcology. In 75 per cent. of the cases the findings of the test coincided with the anatomic findings, but in the other cases they conflicted. He concludes that an entirely negative general and local reaction may be accepted as excluding in all probability a tuberculous process in the genital or urinary apparatus, but that the positive findings must be accepted with some reserve.—*Journ. A. M. A.*, vol. xlix, No. 21.

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TWO LECTURES
ON
DYSENTERY.

Delivered at St. Thomas's Hospital.

By F. M. SANDWITH, M.D., F.R.C.P.,
Lecturer on Tropical Diseases at the Hospital;
Physician to Out-patients, Seamen's Hospital.

LECTURE II.

GENTLEMEN,—I shall take to-day the subject of amœbic dysentery, the geographical distribution of which is somewhat different from that of the bacillary form of the disease. In many parts of Northern Europe, generally in towns, there have been reported sporadic indigenous cases of amœbic dysentery. It is more frequent in the centre and south of Europe than it is in the north, and the disease is not known in this country if we except the imported cases. In the United States this is the commonest variety of the disease, and this is so. too, in the West Indies, Guiana, and Brazil. The disease is endemic in many parts of the world, chiefly in Africa, for instance, in Egypt, Algiers, Senegal, Mauritius, and in many parts of tropical Africa. It exists also in Siam, and in China. Both forms of dysentery are very prevalent in the Philippine Islands. Strong and Musgrave, writing from Manila, reported 561 cases of amœbic dysentery and 766 cases of bacillary dysentery. There are many parts where the amœbic disease exists but rarely, such as Ceylon, some parts of India and South Africa.

With regard to etiology, insanitation is the most common factor in the predisposition to this disease, in other words want of hygiene, and included in that there is the impurity of the drinking water supply. Soil and water are the two natural homes of ordinary amœbæ, and in warm climates amœbæ are constantly found in surface water, cisterns, tanks, and even in the pipe water of towns. Dysentery occurs often after floods of rain, not only from the chill which the patient says he feels and which he believes is the cause of the disease, but from the surface water which he drinks which has become contaminated. It is difficult to understand exactly the pathology of chill, but probably the chill acts on the phagocytes and the normal intestinal flora in some way which prevents the invading amœbæ being destroyed; they then flourish and colonize, and that is why we get the dysentery occurring after a chill. Probably the chill has no more to do with it than in the case of pneumonia, which we believe to be due to the invasion of the pneumococcus. Raw salad and vegetables are dangerous conveyers of dysentery, especially in countries where liquid human sewage is used for manuring gardens. Dysentery has occurred in Europe in barracks which have been previously occupied by dysenteric patients. So you see people are capable of bringing dysentery from an endemic centre into a country, like this, where it does not naturally exist. Then, the hands of the patient, or of the nurse, or the vessels may be soiled by dysenteric fæces, and then come into contact with the mouth of another person and so convey the disease. With regard to the season when dysentery is likely to be rife, we may say that in the true tropics it exists all the year round, but in sub-tropical climates it is absent in the winter and present in the summer. As regards race, there is no immunity for any particular kind of people, but strangers all over the world are more liable to fall victims and to be more severely attacked than those who are residents in an endemic area. Opinions differ as to how far alcohol is a predisposing factor in the production of dysentery. My own experience is, that the immoderate drinker of alcohol is, perhaps, not more likely to get dysentery than the teetotaler, but he is much more likely when he gets it to have abscess of the liver. So I cannot say alcohol is a predisposing factor of dysentery itself, but it is a factor for the chief complication of dysentery. Troops suffer tremendously from this disease, as they do from enteric fever, when they are exposed to the privations necessary to a campaign. You may like to be told something about the history of the discovery of amœbæ in the human intestine. So long ago as 1860 Lambl called attention to amœbæ in the fæces of a child, and some three years later Loesch published the case of a man suffering from chronic diarrhœa in whose fæces amœbæ were found. He also discovered that a weak solution of quinine (1 in 5000) killed the amœbæ when injected into the bowel, though amœbæ returned when the quinine enemata were left off. Death ensued eventually from pneumonia, and the large intestine was then

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found to be the seat of chronic ulceration. Portions of the fæces containing amœbæ were injected into four dogs; three of them remained well, but the fourth animal, in eight days' time, passed some mucous stools containing amœbæ; it was then killed and the large intestine was found to be congested and ulcerated. I now pass over many years in the history and advance to 1883. While Professor Koch was discovering the bacillus of cholera at Alexandria, he found in four cases of dysentery, after death, amœbæ in the intestine, and he was the first to consider that the presence of amœbæ in the deeper part of the intestinal ulcers was indicative of the real cause of dysentery. Kartulis, a Greek doctor, in Alexandria, stimulated by Koch's enthusiasm, as, indeed, we all were, then investigated many cases of dysentery and noted a relation between the numbers of the amœbæ and the severity of the symptoms. He also succeeded in inducing dysentery in a cat by injecting into its rectum some fresh dysenteric fæces. The animal's stools contained amœbæ and the cat died on the twelfth day. Many confirmatory reports of the connection between amœbæ and dysentery then came from various countries: from the United States, Italy, Germany, Austria, and Brazil. The next step in the process occurred when Kruse and Pasquale found amœbæ in the stools of several healthy persons in Egypt. Some of these pictures are the fruits of their labours. They found amœbæ in their own stools and also in the stools of several other healthy people, from which they concluded that in some countries amœbæ are occasionally normal inhabitants of the intestine. Differentiation was then made between what are now called the amœbæ of dysentery and the non-pathogenic amœbæ coli. Next, Marchoux succeeded in causing dysentery in a series of twenty cats by means of enemata of amœbic fæces, and in those of the animals which lived for fifteen days or longer after the injections abscesses of the liver developed. As a rule there was a single abscess, but on one occasion there were found to be as many as seven small abscesses. If the dysenteric stools were heated to 113° F. for thirty-five minutes no infection took place. This temperature was chosen because it destroyed the amœbæ but not all the bacteria of the fæces. It is now generally conceded that in the human intestine amœbæ are often present, while in tropical and sub-tropical countries a species of pathogenic amœbæ also exists which is capable of producing both dysentery and abscess of the liver, and hence we call this variety amœbic dysentery, to distinguish it from the other variety known as bacillary dysentery. There are at least three species of amœbæ which are internal parasites of the human body. First is the *Amœba buccalis*, which feeds chiefly on leucocytes and bacteria in carious teeth. The second is the *Amœba coli*, which occurs in the large intestine, and has soft, semi-fluid pseudopodia, which are unable to force their way into the epithelial cells of the intestine; this amœba is therefore obliged to feed on bacteria and other substances in the intestine. The third is the important one, named the *Amœba histolytica*. This is armed with tough pseudopodia, and is able to attack the epithelial cells of the intestine, and to penetrate into the submucous tissues, where it causes ulceration, while it feeds on the tissue elements and the blood-corpuscles of the host. Thus there are three amœbæ: one in the mouth; one in the colon, which you need not trouble about because it cannot get inside the peritoneal cavity; and then there is the important one which burrows into and through the mucous membrane and the submucosa and destroys them, producing necrosis, ulceration, and very often a liver like the one I now show you.

Morbid anatomy.—In virulent cases the whole of the large intestine may be involved, but the lesions do not often extend above the ileo-cæcal valve. In at least half the dysentery autopsies which I have seen in Egypt and Turkey the whole of the large intestine has been attacked. In some cases of amœbic dysentery which have died early it is only the rectum which is affected, and, speaking generally, the lesion seems to be more advanced in the lowest part of the large intestine. There are circumscribed areas of thickening and necrosis in the submucosa, giving rise to nodules projecting into the mucous surfaces of the bowel, resulting in a punched-out appearance. These nodules are roundish or oval, and may vary in size from that of a hemp-seed to that of a large bean. When several ulcers coalesce they are found to be transverse to the long axis of the bowel, and they vary greatly in depth, this depth depending on the degree of necrosis present. In mild cases the mucous membrane between the ulcers looks fairly normal to the naked eye. The amœbæ are found in the viscid

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contents of the nodules, and in the surrounding necrosis. It has not yet been quite decided by what method the amœbæ reach the submucosa, but Schaudinn and others consider that they probably enter by Lieberkühn's crypts. There is not much more to say about the morbid anatomy of dysentery when we leave the intestine, but I would remind you that the peritoneum is thickened and discoloured at the various sites corresponding to the deep ulcers. Adhesions are often found in old-standing cases between the coils of the large intestine and the neighbouring parts. You scarcely need to be reminded that perforation may occur in this form of dysentery, as it does in the bacillary variety.

Now, as to the symptoms. The disease may be either acute or chronic. In the acute disease we have pain, diarrhœa, tenesmus, and peculiar stools. At first the stools seem to the naked eye to be normal, so that the patient begins by saying he had only a little diarrhœa, and afterwards he had stools containing a little blood and mucus, and then eventually tenesmus, increase in the number of stools, and their contents became only mucus, and, may be, blood-stained mucus. If gangrene occurs sloughs are passed. There may be small clots of blood in the motions, and the odour is most disagreeable. The quantity of blood is usually not very great, and dangerous hæmorrhage is quite rare, distinguishing it in that way from typhoid fever. The temperature varies between 99° and 102° F., usually a little lower in the morning and higher in the evening. The pulse corresponds with the temperature. The appetite is lost, the tongue at first is moist and slightly coated, and then in dangerous cases becomes dry and brown. In such cases there is marked prostration, out of all proportion to the diarrhœa and the hæmorrhage, and the urine may contain a trace of albumen. In fatal cases death may take place in a week or ten days from exhaustion or from general peritonitis following perforation. In milder cases permanent or temporary recovery occurs at the end of two or three weeks.

Chronic amœbic dysentery.—Most cases run a chronic course unless enemata have been used, that is to say, large enemata to kill the amœbæ. The patient varies between a fairly convalescent state, with symptoms which are attributed to indigestion, and relapses lasting a few days, during which time the motions are more frequent and some blood and a good deal of mucus is passed. By rest and dieting these symptoms disappear until some error in diet, or fatigue, or exposure to cold heralds in another attack. When this condition has continued for some time the general health becomes impaired, the chief symptoms being debility, loss of flesh, anæmia and loss of appetite. The digestion becomes more difficult and the patient finds that he is only happy when taking liquid food. When recovery takes place the amœbæ disappear from the fæces, the patient has only one motion a day, and may even find that constipation requires for a time regular aperients.

The complications of this disease are, in the first place, liver abscess, usually single and of large size, but you must remember that there may be two or three of them. When I was a student here we were taught that tropical abscess was always single, and that if there were several abscesses it was a proof that there was a pyæmic condition. But that is not true, for there may be a few tropical abscesses, though there is usually a single large abscess in the right lobe. Perforation may occur in gangrenous cases, or in those which have not been strictly treated by rest and diet. Strong, from whom I have already quoted, working in the Philippines, met with twelve perforations in seventy-seven post-mortem examinations. In at least half the number of cases the perforation is in the sigmoid flexure or rectum.

Diagnosis.—From what I have already said it will be clear to you that the discovery of the amœbæ in dysenteric motions cannot be considered an absolute proof that you are dealing with a case of amœbic dysentery, for the amœbæ found may be quite harmless protozoa. When amœbæ are found in fresh, warm stools, or when a fragment of mucus is placed on a warm slide, it is well to see if motile forms of amœbæ are present before making a positive diagnosis. Epithelial cells and other things may look like amœbæ, but if under the microscope on a warm slide you see a pseudopodium protruding a new process, you may be certain there is an amœba. And if you find nothing in the fæces brought to you, and you still suspect that amœbæ may be present, it is not a bad idea to pass a catheter into

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the rectum, and then examine some of the mucus which may be in the eye of the instrument. Any intermittent diarrhœa in tropical countries should be looked upon with suspicion, and the fæces should be carefully examined. In countries where such entozoa are endemic, many cases have been unsuccessfuly treated for chronic dysentery which were really due to the presence of ankylostoma or bilharzia worms. The moral of that is, always examine the fæces under the microscope.

The prognosis of this disease should be guarded because of the tendency to relapses or exacerbations after apparent recovery. According to Dr. Andrew Davidson, a most careful observer, the case-mortality cannot be under 20 per cent., and sometimes is much higher, but here I think he must be referring to the acute cases only.

Prevention.—The rules should be those which you would adopt during an epidemic of enteric fever: thorough disinfection or burning of dysenteric stools, the protection of milk and other food from dust and flies, strenuous care of the water-supply, thorough draining of the soil, and surface cleanliness, boiling of all water used for drinking purposes or for washing, cooking, and drinking vessels, avoidance of raw vegetables, salad and fruit, care about diet and excess of all kinds, including fatigue and exposure to damp cold, and care in cleansing the hands or any other part which may have been in contact with fæces or the bed-pan.

Treatment.—The most important thing is rest in bed, for you cannot rest the intestines unless you rest the whole of the body. A cotton-wool pad and flannel belt should be placed on the abdomen and the patient should have milk if he can take it, and most people can. If he cannot take it give various substances, such as albumen water, plasmon, and if there is not too much diarrhœa, beef tea. The stimulants which should be given will depend on the amount of prostration. You would not give stimulants to an ordinary case, certainly not to a child, unless there were great prostration, but then do not withhold brandy, which is a little constipating and therefore the best; it is of no use to give wine if you have good spirits at hand. Poultices sometimes help locally, but opium may be given by the mouth, or morphia hypodermically, if necessary for pain. A small enema of starch and opium after each motion is very comforting in relieving the tenesmus. It often sends the patient to sleep, and he is happy until he is awakened by his griping pain and desire to pass another motion. When you are watching an acute case of dysentery and you find he has a motion every half hour, you begin to think you have achieved much if you can extend the interval from half to three quarters of an hour, and gradually extend it beyond that to one hour, and then to three or four hours. When a man is passing motions all the time you are thankful for any respite between the operations. Sulphate of magnesia or of soda in drachm doses every hour until there is a fæcal motion is, in my opinion, the best treatment for this form of dysentery. I have hardly made use of ipecacuanha since I learnt of this treatment from Dr. John Anderson in 1885. He tells me that he derived it from the text-book on materia medica by Professor Bartholow, of Philadelphia. This treatment of late years has been freely employed in India, South Africa, and elsewhere, and is much practised by those who have found ipecacuanha unsatisfactory. For many years large enemata have been used in chronic dysentery, and I have never been able to understand why the patient should not be given the advantage of this treatment early in the case. The objections to the use of two or three pints of enema are that there is danger in distending an acutely inflamed bowel or of perforation in sloughing cases, and also that the pain and sense of straining sometimes cause the patient to be unable to retain the liquid. But for some years I have given both to adults and children large enemata which have cut short the disease in a very satisfactory way. Small enemata are of no use except to relieve rectal symptoms, because the drug has no chance of reaching the place where the ulceration is. I usually begin with a soothing, cleansing enema of flax and water in quantities of one ounce of flax to two pints of water. This is made like an ordinary infusion by soaking linseed for two hours in warm water, and then mixing it with a larger quantity of warm water at the temperature of the body. I used to give boracic acid 1 in 30 and starch 1 in 40 in a two-pint enema, but though this answers quite well in many people, in some Europeans I have found that boracic acid produces nausea and vomiting, and a scarlet erythematous rash over the face, body, and limbs. There-

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fore I have discontinued it and give in preference linseed enemata, the use of which I learnt in the United States. I generally order this wash-out at night, after which the patient is inclined to sleep. You must of course give all enemata after a motion. The morning should be reserved for the curative enema, which should consist of nitrate of silver or sulphate of copper, in the proportion of 1 in 1000. Again two pints should be employed, and some patients after a time can take three or four pints. About twenty minutes should be occupied in slowly administering the injection by gravitation through a soft rubber tube passed some six inches into the bowel. The foot of the bed should be raised at least two feet, or if that is not possible, the buttocks of the patient must be raised by two or three pillows under him. If in spite of washing the anus after every motion there is some local soreness, it will be necessary to paint the anus with cocaine before the india-rubber tube is introduced. If that is not sufficient a suppository of one grain of cocaine should be inserted into the rectum half an hour before the curative enema. In some exceptional cases I have had to give morphia hypodermically after the use of this enema. The flax enema does not hurt the patient, but the other does, and you never know what amount of irritation you are going to produce, partly because you do not know how well or how badly the patient will stand the pain, and you do not know exactly the condition of the interior. If the ulcers are already raw and irritating, you can easily see that the nitrate of silver or the sulphate of copper, which latter I often use, will produce great pain, though not always. There is a patient I am now seeing in the Home of this hospital who is having three pints every day, and he has begged for another week beyond the time I originally told him he must have the enemata. There are chemical reasons for not adding laudanum to the solution of sulphate of copper or nitrate of silver. Many people use quinine injections of the same strength or even 1 in 500, but though weaker solutions of quinine will kill the amœbæ in the laboratory at once, I have by experience found that the salts I have mentioned are much more likely to cure than quinine alone. In all cases I give, as I told you last time, salicylate of bismuth with or without tannigen and opium every four hours by the mouth, in the hope that some of the bismuth may reach the ulcers. Even if it does not do so it seems to exercise a good action on the stomach and small intestine, which are both sympathetically irritated. I have reasons for thinking that the salicylate of bismuth must get down to the large intestine because I used occasionally to give sub-nitrate of bismuth and I found some of that in the fæces. I have told you that this treatment, that is to say the saline treatment of dysentery, aided by bismuth and enemas, is the best treatment that I know for this form of dysentery; I have told you that it is better than ipecacuanha, and when I say that I mean the ordinary ipecacuanha which we talked about last time, which is given in doses of 20 or 30 gr. There is one form of treating dysentery in the chronic form which is useful, which I have seen given by Sir Patrick Manson in the wards at the School of Tropical Medicine. The patient is ordered to be starved for four hours, having not even a drink of water. At the end of that time a mustard plaster is put on the epigastrium. He is not allowed to move, but must lie flat on his back in bed without a pillow. He is given 15 ♏ of laudanum in water, and after fifteen minutes he takes 30 gr. of ipecacuanha powder, and the nurse has instructions to wipe away any froth which occurs on the patient's lips and absolutely to prevent the patient moving for at least three hours, and during those three hours he is not allowed to have any food. This is generally done at night. Next night a similar routine is gone through, but he only gets 25 gr. of ipecacuanha powder, preceded by 10 ♏ of the laudanum, that is to say less ipecacuanha and less opium. On the third night he gets 20 gr. of ipecacuanha, preceded by 5 ♏ of tincture of opium. The fourth night he has 15 gr. of ipecacuanha without any opium at all. On the fifth night he gets 10 gr. of ipecacuanha, and on the sixth night 5 gr. of ipecacuanha. That is to say, you gradually reduce the dose every night until you get from 30 gr. to 5 gr., proceeding by 5 gr. stages, giving opium for the first three nights only. And that treatment of 5 gr. without laudanum is continued for another week. Amœbæ are found to disappear from the fæces after one or two doses of ipecacuanha or after one large enema. Severe hæmorrhage, as I have told you, is not very common in amœbic dysentery; it is more common in the other form, and is sometimes treated by

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ergotin or adrenalin, or by applications of ice to the abdomen. I have not yet heard of any successful surgical operation in this disease after perforation has taken place. A change of climate is, of course, useful in obstinate or relapsing cases, but this is not nearly so valuable a treatment as that by enemata. Therefore when you have an early case, or even a chronic case, get rid of the diarrhœa and blood in the mucus, kill the amœbæ, and then send the patient off on a sea voyage. But if you send him away with the amœbæ inside him they will go on multiplying, and there is more chance of the ulceration being deeper at the end of the voyage than before it. Change of climate and sea air are a fetish which we have inherited from our ancestors, who did not know anything about amœbæ or bacteriology. They are very valuable allies, but they are not of much use when you are dealing with acute disease. In chronic cases, which come to you with a dismal history of relapses, indigestion, and a feeling of general illness, it is difficult to know how long you should treat them. So far I have had good results after a course of three or four weeks. There was a British Medical Association meeting at Edinburgh a few years ago and I read a paper there on giving these large enemata in cases of dysentery before the chronic stage came on. I had just begun it, and I was anxious to talk about it. To my surprise nearly everyone who spoke on the subject condemned me, and said it was an impossible proceeding and very wrong, and they had not seen or heard of it. But one man got up after a time and said he did not understand why so many people objected to the treatment, because, as far as he knew, it was carried out in America and should be carried out. That man was Professor Osler. I did not mind the other colleagues objecting so long as I had a champion of that kind on my side. I have a note on a case which may interest you as showing the way in which people living abroad may become ill; it illustrates also the way in which illness may be brought to them without their knowing anything about it or being apparently able to help it. An English lady living on the first floor of a house in Cairo suffered from fever, diarrhœa, and sore throat. The diarrhœa became dysentery and it proved to be an ordinary case of that disease. Three months later an English child, æt. 6 years, living in the same house but in the flat higher up, on the second floor, became ill apparently because the cesspool of the neighbouring house, immediately beneath the bedroom window, overflowed so that the yard adjoining the house was swamped with liquid sewage. Seven days after the cesspool overflowed the child had definite dysentery with a temperature of 105° F. The bowels the first day were opened three or four times, and by the time I was called to see the child the bowels were acting every half hour, and nothing but blood and mucus was coming away. She was treated in the way I have described to you, and after fourteen days' treatment she was sent away to a convalescent home and was well. It is possible this was an infection by means of fly-polluted milk. First of all there was the woman living in the house who had an attack, three months later there was a surface swamp of sewage, and then the child got a definite infection which may or may not have been caused by the woman living in the same house. The interesting thing about that series of events is that the people who were living on the ground floor were not affected; I saw the children playing about in the road. The difference was that they were Greek children and they were apparently perfectly healthy. Greeks are not so susceptible to any of these intestinal diseases, including typhoid fever, as Anglo-Saxons are. So those who intend to go far abroad had better look after their intestinal flora more carefully than seems to be necessary in the case of people who are not Anglo-Saxons.

December 23rd, 1907.

Acute Poisoning after Oil Enemas.—Buttersack reports a case of serious poisoning accompanied by methæmoglobinæmia occurring in a woman, æt. 33 years, who was given an enema of 250 c.c. of oil of sesame for an attack of gallstone colic. This remedy had been frequently used before for the same condition without any untoward consequences, but on the occasion in question the injection of the oil was promptly followed by symptoms of collapse, which in a few hours became so severe as to make recovery seem doubtful. The blood was chocolate-coloured and gave the characteristic methæmoglobin spectrum, while the surface of the body was cyanotic and cold, especially the face, ears, fingers, and toes.—*Med. Record*, vol. lxxii, No. 22.

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A CLINICAL AFTERNOON AT THE POPLAR HOSPITAL FOR ACCIDENTS.*

(*Concluded from p.* 160.)

Mr. RIGBY: I show you, gentlemen, a woman of middle age whose history briefly is this: When a young child she had *ascites* and her legs began to swell. During last year she had pain in the heart and in the abdomen, for which she went to St. Bartholomew's Hospital, where she was in the medical wards for some time. Having definite ascites, she was sent over to the surgical side, where laparotomy was performed and the fluid evacuated. Nothing else was done. She then came here under Dr. Theodore Thompson's care with ascites, and she soon began to develop symptoms of intestinal obstruction. He asked me to see her. After tapping one saw that there was definite intestinal peristalsis, which gradually increased; she got a recurrence of the ascites, and finally had acute obstruction. I operated on February 10th and found in the abdomen a large quantity of clear straw-coloured fluid, and an enormous amount of distension of the small intestine. I could not see any large intestine at all. On tracing down this distended gut towards the cæcum one came on a peculiar condition of about six inches of the lower portion of the ileum, a gradual narrowing of the lumen and thickening of the walls, the whole presenting a very anæmic appearance. This narrowing increased until one got to within one and a half inches of the ileo-cæcal valve, where the ileum was very small. The cæcum was quite undeveloped, and the ascending colon looked like that of a child. The transverse colon I did not notice. I am sure the condition was not a growth of any sort, and there were no tubercles to be seen anywhere. The appearance was unlike anything I have seen, either in the post-mortem room or elsewhere. I opened the ileum above this peculiar condition and emptied some pints of fæcal material, and then joined the ileum to the sigmoid by a lateral anastomosis. Since then she has gradually developed a little more fluid in the peritoneal cavity, and you can feel the tumour itself. She gradually got over the trouble, and she is now progressing. I believe the condition to be developmental. I opened the small gut just above this peculiar thickening of the wall and tried to pass a probe down to see if there was membranous obstruction. The probe went as far as I could reach, and I put my finger in to feel the condition of the walls. They felt like much-thickened but otherwise normal intestinal walls. As far as the finger could reach it was evident there was a gradual narrowing of the canal, so that near the ileo-cæcal valve the lumen was very narrow. This obstruction must have gone on for a very long time. The small intestine was so dilated that it was mistaken for colon. It lay in the position of the transverse colon and was of enormous size. With regard to the question of making an anastomosis in acute obstruction, it is not a thing which you like to do if the obstruction is very acute unless you first empty the intestines very carefully. In acute obstruction one gets so much temporary paresis of the intestinal wall that if a lateral anastomosis is made the gut above empties itself insufficiently. It is safer to make an external opening. In this case I closed the abdomen completely. One knows that draining the ileum is a hazardous procedure. At first there was a tendency to diarrhœa in this patient.

Dr. THEODORE THOMPSON: I should like to say a few words with regard to her medical condition on admission. She was a wasted woman, with a distended abdomen, and it looked like a case of tuberculous peritonitis, such as one sometimes sees even in women of this age. The abdomen was very tense and contained much free fluid, and I thought it best to withdraw the fluid and see what was beneath it. After the fluid had been removed one could both see and feel large sacculated coils of intestine, which both Mr. Rigby and myself thought were hypertrophied and distended portions of the colon. There was marked peristalsis, and one could see the waves passing from side to side of the abdomen, but chiefly in the region of the colon. There was also a loud gurgling sound, which could be easily heard at the side of the bed. The purgatives which were given failed to produce any motion. The bowels could only be emptied by means of enemata. The results were large and consisted of properly formed fæcal masses, showing that the obstruction could not be very low down in the large intestine. This was noticed as a curious

* A meeting of the Hunterian Society, with Mr. JOHN POLAND, President, in the chair.

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feature at the time, since the physical signs seemed to point conclusively to obstruction of the sigmoid colon. The mystery was explained when at the operation, the enormously hypertrophied coils were found to be small intestine and not large.

Mr. RIGBY: This boy shows the result of an operation which is not often performed for *stone in the ureter*. When he came in he gave several months' history of pain in the right ureter and kidney with attacks of hæmaturia. There was no penile pain, no frequency of micturition, but fairly free bleeding. A radiograph was taken and showed stone in both the kidney and right ureter. The stone in the lower ureter appeared to be quite close to the bladder. I removed the stone from the ureter by the posterior or paro-rectal route, and I took away the kidney stone at the same time by an incision in the loin. The alternative operation is to prolong the lumbar incision forwards, and if the stone is quite close to the bladder, as this was, namely one inch off, you must make an enormous wound, with a corresponding extensive division of the abdominal muscles. The back route avoids the risk of ventral hernia afterwards, and the damage to muscles in the abdominal wall. In addition to that, drainage is perfect by this route. There is no disability at all. The gluteus maximus becomes somewhat wasted at the lower part where the fibres are cut. But for this route to be chosen the stone must be quite low, not more than two inches from the bladder; otherwise I think it is easier to get at it by the ordinary inguinal route above. I should like to hear the opinions of Fellows about this point, because most surgeons say you should remove stones of the ureter by the inguinal route. I have done this operation now four times, and I am sure there are great advantages if the stone is within two inches of the bladder and not higher up than in this case. There is practically no bleeding, and it is very easy to stitch the ureter, much more so than by the front route. In a man with well-developed muscles this posterior route is not so easy to work on.

Mr. POLAND: Do you completely divide the sacro-sciatic ligaments by that route?

Mr. RIGBY: The steps of the operation are these: you make an incision three and a half inches long, cut through the posterior fibres of the gluteus maximus, then cut through the great and small sacro-sciatic ligaments. Then divide the coccygeus muscle by blunt dissection, and then you have to go through the rectal fascia. This brings you into the sub-peritoneal tissue. You feel for the ischial spine, which is your first landmark, and the ureter is half an inch above that. If you cannot find it at first get an assistant to push down the abdominal wall. If you cannot find it then, work forward to the vesicula seminalis and take that as your guide. There are many points which crop up in the operation itself, which perhaps I need not go into now. One of them is to avoid stripping the peritoneum up too much, otherwise it sags away. Once the ureter is defined it can be more easily dealt with than by the other operation, you can bring it nearer to the surface of the skin and can stitch it easily and without any trouble. The wound leaked urine in this case for a week and then became dry. I show you the scar of the incision which was made.

Mr. OPENSHAW: I agree with Mr. Rigby that in adults it is very difficult to reach the ureter by the rectal route. I have operated upon one or two cases by the abdominal method, and it seemed easy to reach the stone. In one case it is true the stone was not so low down as it appears to have been in this case. There is no doubt that stones do sometimes get wedged in the ureter and can be moved neither up nor down by outside manipulation. Therefore it is necessary to remove them by operation. The last patient I operated upon for stone had had pain for about five years, with attacks of bleeding which regularly incapacitated him, sometimes at a moment's notice. Operation is essential, and the only question to settle is which route it shall be done by. I think we are indebted to Mr. Rigby for bringing these cases forward, and for giving us the rule that the posterior operation should not be undertaken unless the stone is within two inches of the bladder. There is no doubt that a stone in the lower part of the ureter is deep in the pelvis. It is, however, astonishing how easy it is to reach the ureter if a long curved incision be made, as if for the ligature of the common iliac artery. Care must be taken to divide the iliac fascia, otherwise the ureter and vessels are pushed forward out of the operation area.

Mr. OPENSHAW: I show you here a man who

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came for treatment with firm *fibrous ankylosis of both knees*, there being no movement whatever. He was in the London Hospital for seven months. I worked his knees under an anæsthetic several times, and put him into one of the Hessing's splints which I have already exhibited. He has now been at work for twelve months. The movement at each knee is quite free. The case was one of gonorrhœal arthritis with ankylosis, and much other treatment had been tried without benefit.

Dr. THEODORE THOMPSON: This old lady came into the hospital complaining of shortness of breath. She has rather an *unusual form of heart trouble*. At night-time she has very peculiar attacks. She may be sitting up in bed when she suddenly falls back and scarcely breathes. Several times during these attacks the pulse has been examined and could scarcely be felt at all; it becomes very irregular and much slower than usual. The heart is dilated and hypertrophied, and both aortic and mitral regurgitation are present. In addition we find that she has tabes dorsalis. She has had "rheumatic" pains all over her for fifteen years, and her gait has been unsteady for four or five years. The pupils are small in size, and while reacting to accommodation fail to do so to light. The knee-jerks are absent, and there is a marked degree of anæsthesia, confined to the left leg. The interesting point about the case is the association of the heart trouble with tabes dorsalis. Aortic disease is twice as common in subjects of tabes as it is in the general population, and I suggest that the lesion in the heart in this case is a syphilitic aortitis causing ulceration of the aortic valve, this ulceration leading to secondary mitral regurgitation, and that the heart failure has resulted from this. The peculiar syncopal attacks with bradycardia may have been due to interference with the conducting fibres of the heart at the aortic ring.

The next case is that of a man, an unusual case, who was sent to me by Dr. Eccles. He had been ill for three months, having been suddenly taken ill on December 26th with pain on the right side. The diagnosis of influenza was made, and he laid up four days, and then went to work for a week. Then he got short of breath and began to cough up foul sputum. On January 12th, 1907, he had a rigor, and throughout January and the greater part of February his temperature was high, ranging between 100° and 102° F. At the base of the right lung there was found to be an area of diminished resonance over which one could hear crepitations and tubular breathing, and during January he coughed up about a pint of foul sputum every day. We could find neither tubercle bacilli nor streptothrix in the sputum. When he came in one would have said he was almost certainly going to die. He was very emaciated and the pulse was feeble, the first cardiac sound was almost inaudible, and he was coughing up a quantity of this foul sputum. The condition was thought to be gangrene of the lung. I suggested it might be only an abscess of the lung, and I treated it in that belief. He was given creasote, at first in small doses, and as he took it well the dose was increased. Finally he was taking half a drachm of creasote by the mouth in cod-liver oil daily. He also had inhalations of creasote and iodine, with Burney Yeo's inhaler. During the two and a half months of his stay in the hospital he has gained eighteen and a half pounds weight, the physical signs at the base of the right lung have diminished, and the temperature has been normal for three weeks. He still coughs up foul sputum, but only a small quantity daily. I exhibit a radiograph which shows the abscess. He is now going to a convalescent home.

The last case is this girl, æt. 20 years, who is a typical case of that very well defined disease, *disseminated sclerosis*. She has been ill two years with weakness in the left leg. Two years ago she was admitted into another hospital, and was thought to be a case of hysteria. Later she developed the condition you see to-day. You will notice that she has the typical staccato, scanning speech, and the marked nystagmus. All four limbs are extremely spastic, and there is marked ankle clonus. All the deep reflexes are increased, and Babinski's sign is present. I ask her to hold out her hand, and you see the violent intentional tremors which are produced. She has experienced some difficulty in holding her water. Her fields of vision are not greatly contracted, but the optic discs look pale, especially the temporal halves, and I think optic atrophy is commencing there. Her mental condition is one of extreme gaiety, and she laughs on the slightest provocation. Although completely bedridden, she seems perfectly happy and contented. She presents an extremely typical clinical picture of disseminated sclerosis.

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THREE LECTURES

ON

SURGICAL DISEASES OF CHILDREN.*

By A. H. TUBBY, M.S.,

Surgeon to the Westminster Hospital, and Consulting Surgeon Evelina Hospital for Children.

LECTURE I.

GENTLEMEN,—I purpose to day, as you are aware, to speak for a short time on some surgical diseases of children. And the first subject I will take is *asphyxia neonatorum*, which has very far-reaching surgical effects. First of all as to its causation. There are two types of asphyxia occurring, one before the birth of the child, and the other after, but the occurrence of the latter is not so frequent as the former. Among the numerous causes which are given, there is no doubt one which is chief, viz. a feeble and flabby development of the child. The rib cartilages are extremely soft, and it is not possible for the child to carry out properly the movements of inspiration and expiration. Another cause is the ingestion of mucus or amniotic fluid, and rarer causes are to be found in certain congenital developments or mal-developments, not the least of which are syphilitic stenosis of the trachea, pressure on the trachea exerted by enlarged glands, or a mal-developed diaphragm. In some cases there may be syphilitic hardening or syphilitic induration of the lungs. All those conditions give rise, or are likely to give rise, to the condition known as asphyxia neonatorum.

We will now pass on to consider for a moment what are the symptoms more particularly with regard to the child. There are two degrees of asphyxia, the lesser degree or cyanotic stage, and the extreme degree or pallid stage. The first of

* Delivered at the Polyclinic.

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these is known as asphyxia livida, and the second as asphyxia pallida. The former is evidenced by a cyanosed condition in the face and lips, and some loss of tone in the muscles, but not entire loss of such tone, while in the asphyxia pallida there is practically complete loss of tone. The child is seen to be cyanotic in the first stage, but the efforts at breathing show that the asphyxia is not complete. In asphyxia pallida the child is pale, the limbs are toneless, and there is no discoverable attempt at respiration. The first stage of the condition may be recovered from, but the second stage is beyond hope. There are contributory causes of asphyxia, which may be traced to the peculiarity of the fœtal circulation. You, of course, know that in the fœtal circulation the blood of the umbilical vein mingles with the blood of the vena cava and mixed blood passes through the lungs. It is commonly supposed that the child's lungs are inflated very rapidly after birth, but such appears not to be the case. There are observations on record which show that in a small infant who died three days after birth, portions of the lungs were found to be non-inflated, a condition which is known as atelectasis. It is doubtful how long a period is required for the lungs after birth to become completely inflated, but it is certain that the inflation is not a sudden but a gradual process. And the result, of course, is that if the child is very feeble it succumbs very shortly after birth. But I want to speak more particularly of the results of prolonged asphyxia, which are very curious. The most important result of prolonged asphyxia is undoubtedly that very serious one known as cerebral hæmorrhage or apoplexy neonatorum. Its consequences, as I shall tell you presently, are far reaching and extremely deleterious to the child. It is very curious to note in children who are the subjects of asphyxia neonatorum that they not infrequently get jaundice after about the third to the fourth day after birth, and some of them undergo acute fatty degeneration. Others are subject to various hæmorrhages, of which I shall have to speak later. With reference to the treatment of asphyxia neonatorum, if it is of the pre-natal form the proper thing, of course, is to hasten the birth as much as possible. If after birth you should invert the child, and take good care to clear the fauces, and if necessary pass a catheter into the trachea and inflate the lungs. Or it may not be found necessary to do as much; all that is required is to slap the child vigorously, and apply a cold water douche. If these means are not successful, it is necessary to employ the Sylvester method of artificial respiration. But a feeble child should not be left uncared for too long, because very often respiration begins and appears to improve, but if the child is left alone respiration gradually sags away. There is one condition for which asphyxia neonatorum may be mistaken, and that is hæmorrhage. In cerebral hæmorrhage the breathing is good at first, but becomes gradually less and less, especially if blood is effused in the medulla. You will also notice that the fontanelle is not depressed as it is in asphyxia, but is projecting. And in cerebral hæmorrhage you will find signs of paralysis, while, as the respiration becomes slower, the condition deepens into one of coma. So you see the distinctions are clear. In asphyxia there is feeble respiration, which, under proper treatment, will increase in frequency and volume, and there is a depressed fontanelle. But in hæmorrhage there is a projecting fontanelle. But the importance to my mind is not so much in the asphyxia itself as in the very disastrous results which follow.

We pass on now to a more interesting condition, of which some of the consequences are in some cases so far reaching as to last through the whole of adult life. I refer to *apoplexy neonatorum*, or hæmorrhage occurring in infants, as some prefer to call it. This may take place in one of two situations, either from the choroidal plexus, thence into the lateral ventricles and onwards to the fourth ventricle; or more frequently it is seen on the surface. In that case blood oozes from the vessels of the pia mater, and not from the meningeal vessels or sinuses. The extent of bleeding is often very great, and I have seen the whole of one hemisphere covered by a huge clot, but it may be small in amount and just sufficient to cause pressure on an important part such as the speech centre, causing a defect which may last a very long time or be permanent. Of this kind of paralysis I see a large number. A very good instance is that of a little boy whom I saw a day or two ago. He had had paralysis of the left leg and arm, but he recovered, except that his speech centre is still affected. He is now eight years of age, but can only make a few inarticulate noises. Some, however, of these cases recover in every respect.

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As to the causes of apoplexy neonatorum, the most frequent cause is, undoubtedly, asphyxia. Another is the pressure of forceps. Yet another is the moulding of the fœtal head, in which such overlapping of the bones occurs that the small vessels are lacerated. This is particularly the case with that one which supplies the area round the lower part of the fissure of Sylvius, and such is the explanation of the frequency of the affection of the speech centre. The hæmorrhage is often not suspected. In a child the subject of severe cerebral hæmorrhage you see all the symptoms exactly as in a case of ordinary meningeal hæmorrhage in adults. The respiration deepens for a time, and then gradually diminishes, and finally dies away. You will also observe the coma and the protrusion of the fontanelles. A good many children the subjects of severe hæmorrhage fortunately die on the third day or later, but the survivors are often pitable objects, when the effects of cerebral diplegia and spastic paralysis become manifest. Pressure is followed by a softening, and when this passes away cicatrisation ensues with destruction of the cerebral cells and interruptions in the course of the nerve trunks, so that later in life we see those peculiar athetotic movements which are very typical of the worst forms of diplegia.

With regard to treatment, there is very little to be done. The measures to be adopted are rather preventive than curative. An ice bag to the head may be useful. I am not aware that any operation has been successful in arresting the hæmorrhage and removing the clot.

I now pass to the consideration of *cephalhæmatoma*, which is a condition comparatively frequently met with in children, and sometimes constitutes a source of grave errors in diagnosis. It is seen in four situations: viz. between the aponeurosis and the periosteum of the vault of the skull, and then it is known as sub-aponeurotic; or between the periosteum and one of the parietal bones, and it is then called sub-periosteal; or underneath the dura mater, and is then called the sub-arachnoid form; or it may occur on the surface of the brain, in which case it is cerebral apoplexy. But the most frequent form is undoubtedly the sub-periosteal. And it is the frequency of this variety and the fact that the hæmorrhage is limited by the outlines of the parietal bones that assists the diagnosis. The appearance presented by cephalhæmatoma is quite typical. You will find a fluid tumour corresponding almost exactly to the shape of one of the parietal bones. And the reason is as follows: You know that where the two edges of the bones come close together the periosteum always sends small processes inwards, which become continuous with the dura mater. So each parietal bone is enclosed in its own fibrous capsule, periosteum above and dura mater below. At first the swelling fluctuates, it is painless, and is not at all hot, and it is seen on the child's head almost immediately after birth. If you watch the course which that takes it is somewhat striking. After a few days a certain amount of absorption of the fluid parts of the blood takes place, more in the centre than at the sides, and the swelling is crateriform. The sides of the crater become harder, undergoing fibroid change, and ossification may take place in the blood-clot which is not absorbed. The usual mistake which is made is to presume that a depressed fracture has occurred, or that there is partial deficiency in the skull, with an isolated Wormian bone in it. The treatment of cephalhæmatoma is simple, and that is to leave it alone. Many surgeons have advocated that you should evacuate the blood and let things settle down, but there is one great risk in doing so. You know that small apertures are found in the bone, through which blood-vessels pass and communicate with those immediately beneath. And if by any chance sepsis occurs, you certainly run a very grave risk of setting up meningitis which will end fatally. Another cause of cephalhæmatoma which I have not yet mentioned is the pressure of forceps. The last well-marked cephalhæmatoma I saw was not limited to the parietal bone, but extended downward in a long ridge from the vertex, and terminated between the eyes. Unfortunately the child when seen was two years of age, and the long ugly ridge was of bony hardness. In addition to cephalhæmatoma on the vertex you meet it in other places, such as on the occipital bone.

We must now pass to the consideration of hæmorrhage into the sterno-mastoid, the consequences of which are very far reaching and very important if considered in the light of the causation of congenital torticollis. Of hæmatoma of the sterno-mastoid I have seen a comparatively great number, and in all stages, and I have been interested in tracing what may be called the life-history

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of these cases. The usual cause of hæmorrhage in the sterno-mastoid is difficulty in labour. And three fourths of the cases are undoubtedly associated with breech presentations, the remaining fourth being associated with vertex presentations. The muscle which usually suffers is the right sterno-mastoid. Rarely is the left affected, but I have just seen a small baby nine months old whose left sterno-mastoid is affected. Presentation was vertex, and occipito posterior. It is evident that the hæmorrhage is due to stretching of the sterno-mastoid in the effort to deliver the head. What is noticed, perhaps even a few days after birth, or sooner perhaps by an observant nurse, is that in one of the sterno-mastoids, usually the right, there is an oval, fluid swelling. And if the further history of this swelling is traced it is found that the swelling becomes less after a time but does not disappear, and is replaced by a more or less solid tumour composed of ruptured muscle fibres, blood-clot and fibrous tissue. In some cases the cicatricial processes extend widely. And I remember seeing with the late Professor von Volkmann a case in which the whole muscle was replaced by fibrous tissue of cartilaginous hardness. More particularly with regard to the life-history of torticollis, I was at considerable pains some years ago, when seeing out-patients at the Evelina Hospital, to trace out the history of a number of these cases. In a moderate number of cases, somewhere between one third and half, a hæmatoma was followed by the onset of torticollis. In a small proportion of cases the hæmatoma disappeared and was not followed by any deformity, at least, for as long as I was able to observe the cases, and that was over some five years. In a third group of cases in which congenital torticollis was met with there was no history of any hæmatoma, nor of breech presentation, nor of very prolonged parturition. In a fourth class of cases there was again no history of hæmatoma, but there were clear signs of congenital syphilis. So that in summing up the causes of congenital torticollis one must remember that there are at least two causes, and possibly more. One main cause is hæmatoma of the sterno-mastoid, and a less frequent cause is congenital syphilitic myositis, which is apt to occur particularly in three muscles: the sterno-mastoid, the biceps, and in the rectus abdominis.

With regard to the treatment of these cases, it is difficult to lay down any definite procedure. You cannot lay open the muscle and turn out the clot. As a matter of fact we do not advise, because we know that in some cases the clot is absorbed, and about the sixth or eighth month the tumour disappears. And so it is usual to wait. When dealing with these cases I advise you to be guarded in your prognosis, especially as to the possibility of torticollis ensuing. Some cases in which there has been tumour, where the head is deviated and rotated, are not followed by permanent torticollis if gentle but assiduous movements of stretching the affected sterno-mastoid are carried out. I would therefore say do not be too hasty in condemning a child to torticollis who is the subject of hæmorrhage into the sterno-mastoid or of specific myositis, and do not speak too early of operation. In many cases a mild degree of contraction will be overcome by manipulation and passive movements, and in other cases it seems that much of the contraction passes off spontaneously without any interference whatever. No question of surgical interference should be entertained in such a child under the second or third year of its age.

We must now speak of *obstetrical paralysis*. And it is astonishing to note how prone the public are to blame the medical man for these paralyses. As I mentioned here yesterday when dealing with another subject, it is well known that a most distinguished personage is the subject of this form of paralysis, for which the attendant physicians are unjustly held responsible. The causes of obstetrical paralyses are possibly three. One of the most important, as I have said, is cerebral hæmorrhage, and it gives rise to a condition known as infantile spastic paralysis or Little's disease, together with the more advanced condition of cerebral diplegia and cerebral hemiplegia. Another cause is the pressure of forceps. The public think that this can and ought to be avoided, and they are often very severe with the medical man who was present at the birth, believing his want of skill to be the cause of the trouble, which is, of course, absolutely erroneous and unjust. Still, it is astonishing what an amount of unavoidable damage forceps will do sometimes. Mr. Sydney Stephenson, at a meeting of the Society for the Study of Disease in Children last year, showed a case which impressed me very much. You could see across the cornea quite

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distinctly the scarring which had arisen from the blades of the forceps. In another case paralysis of the facial nerve and brachial plexus followed after pressure of forceps. Paralysis of the brachial plexus follows traction in the axillæ in delivering the child, and in that awkward condition in which the arm of the child is behind the shoulder, and in bringing the arm down the clavicle must necessarily be liable to fracture. The forms of paralysis, apart from spastic paralysis, which are easily seen are those affecting either the facial nerve or the brachial plexus. Of those the affection of the brachial plexus is very much more severe than that of the facial nerve. It is very fortunate that in most of the latter cases of facial nerve injury due to forceps recovery takes place, so that the prognosis in that condition is good. But it is very different in the case of paralysis of the brachial plexus. The local or morbid conditions which are found are as follows. It has been shown by dissection that there may be hæmorrhage or scarring round the roots of the nerves and their branches, forming the brachial plexus, and in such a condition the prognosis is moderately good. More frequently the fifth cervical nerve, or sometimes the sixth, is either torn away from the cord or completely lacerated. The fifth and sixth cervical nerve roots are particularly liable to injury, because they are stretched across the corresponding transverse processes. The particular type of paralysis which arises from laceration or injury to the fifth or fifth and sixth nerve roots is known as the Erb-Duchenne type. The arm is held close to the side, and the patient cannot raise it because the deltoid is paralysed. The forearm is in a state of extension on the upper arm, on account of the paralysis of the biceps and the brachialis anticus, and it is frequently pronated because the supinator longus is useless. The muscles affected are the supra- and infra-spinatus, the teres minor, the deltoid, the brachialis anticus, the biceps, the supinator longus, and extensor carpi radialis longus. An arm so affected has lost two of its main attributes—free movement at the shoulder and free movement at the elbow. With regard to the prognosis of these cases, a certain number of them undoubtedly do recover by massage, and galvanism. The immediate treatment in such a case is, for the first three weeks to leave it alone. It is of no use beginning to massage, to galvanise or otherwise irritate the parts. Your object is to obtain absorption of blood-clot, and therefore as little scarring as possible. During the first three weeks it should be merely placed in a position of rest. Afterwards you may use galvanism, gentle passive movements, and massage. If after two or three years of such treatment you find there is no improvement, and that the child must have a useless upper extremity, you must consider the question of surgical interference. There are two kinds of surgical interference which may be used in these cases. One of them I have described to you before, namely, muscle grafting, but its possibilities are limited, and the best result you can obtain from the muscle grafting is to restore the power of raising the elbow by grafting the outer head of the triceps into the biceps. But so far I have never obtained the power of abducting the upper arm from the side, because it is impossible to make a decent deltoid. Another surgical procedure is nerve grafting, and I think Kennedy, of Glasgow, was the first to cut down on the fifth and sixth cervical nerve roots. He found them torn across, and the ends embedded in cicatricial tissue. He refreshed the ends and sewed them together, and the result was satisfactory. That I consider to have been a most brilliant operation. In the other cases, where this is not possible because of the spaces between the two ends being too great, you must consider the possibility of nerve grafting, and the nerve grafting which offers the best opportunity and gives the best result is to separate the fifth and sixth cervical nerve roots, and embed them in the seventh, and wait until the nerve continuity is gradually re-established, and until the fibres are regenerated, as in many cases happens, from the centre. If you take care to avoid gravity-deformities in the arm, you will find the power of the muscles is gradually re-established.

I think it will be convenient to cease at that point for the present. Next time I will deal with the question of hæmorrhage occurring in the newly-born, and other kindred subjects, such as jaundice.

[For much of the information contained in these three lectures I am indebted to Ashby and Wright's admirable book on diseases of children.]

December 30th, 1907.

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PNEUMONIA AND ITS COMPLICATIONS.*

By HECTOR MACKENZIE, M.D., F.R.C.P.,
Physician to St. Thomas's Hospital and to the Brompton Hospital.

AND DISCUSSION THEREON BY

Mr. A. G. R. Foulerton.
Dr. W. P. Herringham.
Dr. W. Pasteur.
Dr. W. J. Hadley.
Dr. N. Dalton.
Dr. A. M. Gossage.
Dr. A. C. Latham.
Dr. H. A. Caley.
Dr. S. West.
Dr. N. I. C. Tirard.
Dr. J. Fawcett.
Dr. A. B. Harris.
And Dr. W. L. Brown.

For the inaugural meeting of this the Medical Section of the new Royal Society of Medicine, it would not have been possible to have selected at the present time any subject for discussion of greater interest and importance than that of the complications of pneumonia and its treatment by means of serums and vaccines. It is needless for me to dwell on the ravages caused by pneumonia, a plague at the present time more fatal and disastrous to our own population, as I have elsewhere pointed out, than typhoid, diptheria, smallpox, measles, and scarlet fever all combined, and responsible for a large proportion of the deaths at the two extremes of life. It differs from these specific fevers inasmuch as it only rarely appears in an epidemic form, does not generally seem directly communicable from one person to another, and depends on the invasion of the body by a micro-organism which usually appears to be innocuous and is almost constantly to be found present in the secretions lining the mucous membranes of the mouth and nose and other parts of the upper respiratory tract.

Pneumonia, in fact, is due to a parasite which ordinarily we do more than tolerate, to which we habitually afford house and home, and with which usually our relations may be said to be of a friendly character. A man's most deadly foes, however, are those of his own household, and this is certainly the case with regard to those parasites, the pneumococcus and the *Bacillus coli*, which are always with us. From our open enemies, such as the bacilli of typhoid, diphtheria, cholera, plague, tubercle, etc., we can and do adopt preventive measures, but what have we done so far to protect ourselves from these secret and still more deadly enemies which are always at our doors? There is much truth in the words of the old Venetian

"Di chi mi fido guardami Iddio
Di chi non mi fido guarderò Io."

The pneumococcus is a micro-organism which must not be treated as if it were ever harmless, but as one which is always dangerous and against which we ought to adopt preventive measures.

The pneumococcus is a strict parasite and does not appear to be able to flourish apart from an animal body except in culture mediums. It is evidently an air-borne parasite as it is the surfaces of mucous membranes most accessible to the air which it infests. The proportion of normal individuals in whom the pneumococci are found to be present has been generally given as from 15 to 20 per cent. for the mouth and 24 per cent. for the nose. But the latest investigations of the Medical Commission in New York* put it still higher, Buerger having found them in the mouth of 39 out of 78 normal individuals, or 50 per cent., while Duval and Lewis isolated them in every instance from the saliva of healthy individuals. Hiss says it is more than probable that practically every individual acts as a host to organisms of true pneumococcus type at some time or other, at least during the winter months, and probably at repeated intervals. The organisms leave the body with the saliva or sputum, and in either moist or dried sputum may remain virulent for a considerable time. Pneumococci have been found by various observers† in the dust of hospital wards and other places. It is probable that it is through the objectionable habit of spitting that the pneumococci become so generally disseminated, especially during the winter months. Spitting in all enclosed spaces should be forbidden as much on account of the pneumococcus as of the tubercle bacillus.

When we consider how frequently the pneumococcus is harboured in the mouths of normal individuals it is clear that ordinarily either its virulence is low or the resistance of the body is

* An address delivered before the Medical Section of the Royal Society of Medicine.

* 'Journ. of Exper. Med.,' 1905, vol. vii, No. 5.

† Washbourn, "Croonian Lectures," 'Lancet,' vol. ii, 1902, p. 1440.

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high. Some authorities have said that there is no difference to be observed in virulence between a culture obtained from the mucous membrane of a healthy person and one from a case of pneumonia or other pneumococcic infection. Eyre and Washbourn* and Park and Williams,† however, found that cultures from cases of pneumonia were more virulent from rabbits than those from normal persons. The observations of Longcope and Fox‡ suggest that there is a seasonal variation in the virulence of the pneumococci, in the mouths of healthy individuals virulent forms being more common during the winter than during the late spring.

It appears to be more than probable that naturally there is a considerable degree of resistance or immunity inherent in the human body, and it helps us to understand the nature of the disease and its complications if we consider some of the results of the attempts which have been made to produce pneumonia experimentally in animals. The recent investigations of Dr. Wadsworth§ in this direction are of great interest. Previous experimenters, whether they introduced the pneumococci into the animal's body subcutaneously or intra-venously, uniformly failed to produce pneumonic lesions in the lungs. In the case where the virus was introduced by inhalation or by injection into the trachea, occasionally and exceptionally pneumonic patches were produced, but more commonly the lesions resembled those of broncho-pneumonia.

Dr. Wadsworth, however, by previously immunising rabbits and then introducing highly virulent pneumococcic cultures by tracheal injection, directing them as far as possible into one lung, succeeded in producing the typical lesions of acute lobar pneumonia. In producing pneumonic lesions experimentally there were two factors which had to be nicely balanced, the virulence of the cultures and the susceptibility of the animal. Dr. Wadsworth first tried the effect of diminishing the virulence and increasing the susceptibility, but the results were uncertain and unsatisfactory and the animals as a rule died from acute bacteriæmia. Where cultures of low virulence were introduced intra-tracheally in only one out of three rabbits rendered highly susceptible did he succeed in producing typical pneumonic lesions. When he lowered the susceptibility and increased the virulence the case was altered. Out of eleven rabbits which he had previously immunised none died as the result of injecting into the trachea highly virulent cultures, but a few were rendered seriously ill for from twenty-four to thirty-six hours. When killed at the end of three and four days respectively they were all found to have typical pneumonic lesions. Out of five control animals not previously immunised but similarly treated, three died within forty-eight hours without obvious lung lesions while the other two had atypical lung lesions.

These experiments support the view which after a careful survey of the subject I am inclined to take, that pneumonia is a local infection of the lung which the virus reaches through the respiratory tract, there usually setting up a strong local reaction, a defensive process which is Nature's way of preventing if possible an acute bacteriæmia. Locally the micrococci multiply and thence make their way into the blood, for it would seem that a certain degree of bacteriæmia always does occur. In the majority of cases, however, the leucocytes of the blood are able to prevent the body from being overwhelmed by the bacteria.

The bacteria cultivate themselves or maintain their existence for a time in the lung, which appears to be for them a region of what Sir Almroth Wright has called lowered "bacteriotropic pressure," a region where anti-bacterial substances are either absent from the tissue fluids or are there contained in diminished quantity. The bacteria which are carried from the lung into the blood stream do not as a rule succeed in maintaining their existence there, but they may be carried to almost any part of the body and find a footing there. In the great majority of cases the battle between the body tissues and the pneumococci is fought to a decisive finish in the lungs, terminating in recovery or death. Where the anti-bacterial substances are plentiful as in the young, recovery is the usual termination; where they are deficient, as in infancy and after middle life, death is extremely common.

The recent investigations of Rosenow* are

* 'Lancet,' 1899, vol. i, p. 19.

† 'Journ. of Exper. Med.,' *loc. cit.*

‡ *Ibid.*

§ 'Amer. Journ. of Med. Sci.,' vol. cxxvii, 1904, p. 851.

* 'Journ. Infec. Dis.,' March, 1904, p. 280.

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confirmatory of Prochaska's* observations as to the almost constant presence of the pneumococcus in the blood in cases of pneumonia. Rosenow found the microbe in the blood in 132 out of a total of 145 cases in which he took cultures, while Prochaska found it in every one of 50 cases investigated. It is therefore clear that the resistance of the body, especially during the period of life at which the mortality is low, is very considerable.

In the case of all the complications except those in or near the lung, the microbes reach the organs or tissues, in which they set up inflammatory changes through the blood current. The occurrence of complications at a considerable interval of time after the primary lesion is explained by the fact that, as shown by Tizzoni and Panichi,† in the case of immunised animals it is possible for pneumococci to remain in the circulating blood for days, weeks, and even months. The toxine produced by the bacteria is probably the cause of the graver constitutional symptoms, and the crisis is the result of the neutralisation of the toxine by an antitoxine produced by the body. Attempts, however, to obtain the toxine artificially from cultures have not been successful, in fact, according to Welch ‡ it is bacteriologically impossible to do so, nor has it been possible to produce a serum having antitoxic properties. Animals can be successfully immunised, but their serum is anti-bacterial, not antitoxic. It is on this account that serum treatment of pneumonia and its complications has hitherto been so unsatisfactory, so uncertain and so inconclusive.

It is the complications of pneumonia to which we have specially to direct our attention this afternoon, and by far the most common of these is pleurisy, and the most important is empyema. Taking all cases of empyema we find that the pneumococcus is the cause of the disease nine times out of ten, the other organisms which account for the remainder being chiefly streptococci, staphylococci and tubercle bacilli. In sixty-nine cases of empyema in children reported by P. Stanley Blaker,§ a bacteriological examination was made, and in all but four the diplococcus was found, and in only four cases was it associated with other micro-organisms. In a series of 2440 cases pleurisy with serous effusion was observed fifty-four times and empyema twenty-eight times. One case therefore in forty-five had pleurisy with effusion and one in ninety had empyema.

A point about empyema which it is difficult to explain is its greater frequency on the left side than on the right, while pneumonia itself is more common on the right side. One would have expected that right-sided pneumonia would as frequently be followed by empyema as left-sided. Empyema may occur on the side opposite to that of the pneumonia, but it is certainly most common on the same side. Speaking from my own experience I should have said that a pleural effusion after pneumonia is far more commonly purulent or sero-purulent than serous, and therefore I only give the figures I have mentioned for what they are worth. I am accustomed to tell my students that the history of the illness is the most important point of distinction between serous and purulent effusions. If there be a history of illness with an acute onset the effusion is probably purulent and pneumococcic, if there be a history of gradual onset the effusion is probably serous and tuberculous.

Usually the illness due to the empyema is separated from that due to pneumonia by an interval of a few days. Empyema should always be thought of when after the time of the crisis pyrexia continues or returns. When an empyema has been successfully diagnosed it should be treated by incision with resection of rib and drainage. The most difficult cases of empyema to discover are those where there is a loculated collection of pus. Except, however, where the empyema is situated between the lobes or between the lung and the diaphragm it is not a difficult matter to locate the abscess.

With regard to pleurisy with serous effusion I cannot recall any case of an effusion complicating pneumonia which I considered large enough to tap which proved to be serous. The fluid has always been either sero-purulent or purulent. I note that at St. Thomas's no case of pleural effusion requiring aspiration has been recorded in the ten years included in the statistics.

Gangrene of the lung was observed three times

* 'Deutsch. med. Woch.,' 1902, p. 372.

† 'Centralbl. f. Bakter. u. Parasitkunde,' 1905, Bd. xxxvi, p. 25.

‡ 'Med. News,' April 23rd, 1904.

§ 'Brit. Med. Journ.,' vol. i, 1903, p. 1200.

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among 2440 cases. It is fortunately a rare complication for it is almost invariably fatal. When it occurs, prostration, dyspnœa and high fever are usually present. Fœtor of the breath and fœtor of the sputa are usually, but not always, present together, and are generally intense and intolerable. The sputa are always diffluent, often of a dirty brown or chocolate colour, sometimes like pure blood and sometimes of a greenish-yellow colour. The amount of the sputa varies in different cases. Death usually occurs from the fourth to the fourteenth day after the first appearance of signs of gangrene. When the gangrene is diffuse, as it generally is, recovery is scarcely possible, but in circumscribed cases the gangrenous mass may soften and end in an abscess which may be surgically dealt with.

Abscess of lung is according to statistics an even rarer complication than gangrene, of which it is sometimes the termination. It is generally single, and of small size, very seldom larger than an orange, and usually consists of an irregular ragged cavity with purulent, sometimes necrotic, contents in the midst of softened tissue, but in rare cases it may be bounded by a thickened wall lined with pyogenic membrane. The diagnosis of abscess of the lung is a matter of considerable difficulty. There is usually fever of a hectic type, fever which is higher and more persistent than that of delayed resolution. The physical signs are generally ill defined, and only after careful and repeated examination is there found to be a patch of localised dulness with absent or diminished breath sounds, together with local tenderness on firm pressure with the finger. It is significant when one hears friction over the area of local dulness and tenderness. In cases where abscess has followed gangrene fœtor of breath is present. Sometimes abscess of lung is not recognised until it is discharged by rupturing into the bronchi, when a considerable amount of pus is suddenly expectorated with abatement of pyrexia and improvement in the general condition. A white cell blood count may be a help, for abscess of lung would be unlikely in a case in which there was no leucocytosis and no excess of polymorphonuclear cells. Abscess of lung is nearly always fatal, and most frequently is only discovered at the autopsy.

Chronic pneumonia is an unusual and quite exceptional termination of acute pneumonia. After the acute stage has passed away a moderate degree of pyrexia persists, cough continues with mucous or muco-purulent expectoration, there is persistent shortness of breath with probably some pain in the side, and the physical signs of consolidation do not disappear. The symptoms gradually diminish but cough with scanty expectoration and some dyspnœa remain, and it is usually some months before complete recovery ensues. These cases closely resemble chronic pulmonary tuberculosis and should be treated on similar lines. The absence of tubercle bacilli in the sputum and a normal tuberculo-opsonic index are important points of distinction.

Pericarditis is the next most frequent complication after pleurisy. It was observed in 54 among 2114 cases and of these 43 proved fatal. Infection of the pericardium is probably more commonly carried by the blood stream than by simple extension from the lung. This complication is attended with a very high rate of mortality, and only a small proportion of those cases in which pericarditis is recognised during life recover. In a large proportion of the cases the existence of pericarditis is only discovered at the autopsy. Out of 31 cases of pericarditis among 665 cases of pneumonia reported by Chatard* in only 13 was the condition diagnosed. Of the 31, 2 recovered and 29 died. In 19 the pneumococcus was isolated from the pericardial exudate. In only 3 cases was the effusion at all plentiful. It was fibrino-serous in 9, fibrino-purulent in 10, fibrinous in 8 and purulent in 2. The recognition of pericarditis is at present more important from the point of view of prognosis than of treatment, but looking at it as a sign of serious blood infection, I consider that vaccine treatment may possibly prove beneficial in these cases.

Occasionally pericarditis leads to pyo-pericardium, and this complication should be borne in mind, especially in cases in which the presence of affection of the pericardium has been recognised. The area of cardiac dulness becomes greatly increased upwards and outwards and the heart sounds at the same time are muffled and feeble. Pyo-pericardium should be treated by incision and drainage, but even then recovery is the exception.†

* 'Johns Hopkins Hospital Bulletin,' October, 1905.

† See cases reported by Knowsley Sibley, 'Brit. Med. Journ.,' vol. i, 1903, p. 1192; and Fawcett and Steward, 'Clin. Soc. Trans.,' vol. xxxiv, 1901, p. 46.

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Preble, in an important paper* to which I am much indebted, gives the frequency of endocarditis as about 1 per cent.; 126 cases of endocarditis were noted among 11,243 cases of pneumonia. The proportion among fatal cases was higher, 86 cases among 1775, or nearly 5 per cent. Out of 1575 cases I have collected there were 16 cases of endocarditis, and out of 474 fatal cases 13 of endocarditis. One out of every four cases of ulcerative endocarditis is due to the pneumococcus. It would seem that the aortic valve is specially likely to be involved, for it was affected in over half of the recorded cases. The tricuspid valve was affected in 10 per cent. of the cases. The largest proportion of the cases occur over the age of thirty, and pneumonia is more likely to be complicated with endocarditis in the female than in the male.

There is seldom evidence of endocarditis until after the fever of pneumonia has passed off. In the case of empyema an interval of apyrexia follows the primary pyrexia. This interval, however, usually lasts only a few days. The symptoms of endocarditis are as well or as little defined as in cases arising from other causes. It is important to bear in mind that the physical signs of endocarditis may be absent. Endocarditis should be suspected when after pneumonia a remittent type of pyrexia persists without there being evidence of lesions in other organs or parts of the body to account for the temperature. The probability is increased if there is marked anæmia. The appearance of a diastolic murmur or a tricuspid murmur or a systolic murmur conducted towards the axilla would confirm the diagnosis. Sometimes, however, it is not until the detachment of emboli that it is possible to make certain. In some cases endocarditis is not suspected previous to death. The discovery of the pneumococcus in the blood in doubtful cases is important confirmatory evidence.

The prognosis of pneumococcic endocarditis is extremely grave. Among Preble's 132 cases there were but four recoveries, and in three of these the diagnosis was incomplete. Meningitis is very commonly an associated complication. At present one's hopes must rest on vaccine or serum treatment. A case of recovery after serum treatment has been reported by Dr. Newton Pitt.†

* 'Amer. Journ. Med. Sci.,' 1904, vol. cxxviii, p. 782.

† 'Med.-Chir. Trans.,' vol. lxxxiv, 1905, p. 101.

Pneumococcal peritonitis perhaps arises more commonly through primary infection of the peritoneum than as an affection secondary to pneumonia. It is likely, however, that in some cases the primary pneumonia has been overlooked, especially when one remembers how frequently in children pneumonia is ushered in with abdominal symptoms. I have seen cases of acute pneumonia which have been sent up to hospital as cases of appendicitis or intestinal obstruction, or even as peritonitis, without there being any real abdominal affection. When, therefore, pneumonia early becomes complicated with peritoneal infection the probability of the primary lesion not being recognised is much greater. Pneumococcus peritonitis has hitherto been much more frequently met with in children than in adults, and in the female sex it is more common than in the male.

Where the disease is primary in the peritoneum the path of infection is probably through the alimentary canal. Ghon* has reported cases where in peritonitis secondary to perforation of stomach or gastric carcinoma the pneumococcus was found once in pure culture. The pneumococcus does not flourish in an acid medium, but where the natural acid is deficient or absent in the stomach, as in carcinoma, the conditions are more favourable. Where there is gastro-intestinal catarrh the conditions may also be more favourable, due as suggested by Bryant† to the altered condition of the secretions. The constitutional disturbances at the outset of pneumococcal peritonitis are similar to those of pneumonia. Herpes labialis, which is uncommon in other forms of peritonitis, is sometimes met with. I do not think it is likely that infection takes place by way of the genital tract, a suggestion put forward to explain the more common incidence in the female sex.

Bryant‡ has pointed out that the fluid in pneumococcal peritonitis is generally different to what is found in a streptococcus peritonitis. The fluid is thinner than in the case of a streptococcus peritonitis, resembling pus which has been diluted and separating into two layers on standing. In Sir Dyce Duckworth's case§ it was described as thin, turbid, and dark-coloured. In Dr. Frederick

* 'Wien. klin. Woch.,' 1904, xvii, p. 267.

† 'Brit. Med. Journ.,' vol. ii, 1901, p. 767.

‡ *Loc. cit.*

§ 'Clin. Soc. Trans.,' vol. xxxvii, 1904, p. 83.

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Taylor's,* as partly clear and partly greenish-white mucoid pus. In one of Dr. Michell Clarke's† two cases, however, the pus, which was large in amount, is described as greenish-yellow and fairly thick. In the other it is noted that two quarts of thin greenish-yellow fluid were discharged. The prognosis in pneumococcal peritonitis is better than in most forms of peritonitis.

Otitis, although not infrequently the result of pneumococcus infection, is a very unusual complication of pneumonia; only one doubtful case has been observed in ten years at St. Thomas's. The pneumococcus gave rise to otitis media in 18 out of 1449 recorded cases.

Pneumococcic meningitis may appear as a primary disease as well as complicating pneumonia. It is usually acute and purulent and is almost invariably fatal. Death may occur within sixty hours after the first appearance of symptoms. A chronic and sero-fibrinous form of pneumococcus meningitis has also been met with. Meningitis has only once occurred as a complication of pneumonia in ten years at St. Thomas's.

A case of cerebral abscess occurring six months after acute pneumonia was recorded by Dr. Batten.‡ The patient had been ill for many weeks following the attack of pneumonia, and there was a suspicion of empyema, but no tapping or operation had been performed. The abscess contents were very putrid and staphylococci as well as pneumococci were present.

I have had no personal experience of localised neuritis after pneumonia. Daireaux§ says paralyses due to this cause usually implicate the arm on the same side as the pneumonia. He describes also a diffuse form of neuritis resembling diphtheritic, occurring several weeks after recovery from the primary disease. There is first paresis of the lower limbs, and subsequently the arms are similarly affected. In other cases there is atrophy as well as weakness of the muscles. There is no tenderness in the course of the main nerves, and except for occasional twinges of pain no sensory disturbance. Daireaux attributes these cases to anterior poliomyelitis. The same writer has described transitory attacks of hemiplegia occurring in patients suffering from or recovering from pneumonia, which he considers to be the result of some toxic affection of the cells of the motor cortex.

Arthritis as a complication of pneumonia was recognised long before the discovery of the pneumococcus. Grisolle* in 1864 described four cases of arthritis occurring either during or shortly after an attack of acute pneumonia. He noted that the joint affection was persistent instead of fugitive as in acute rheumatism, and he concluded that it was quite distinct from the latter. No doubt many cases of the kind have been observed, but until the discovery of the pneumococcus it could not be actually proved that arthritis was anything more than an accidental association. It is to Weichselbaum† that the credit belongs of having in 1888 first established pneumococcic arthritis as a clinical entity, and since then many cases have been described in literature. The most complete accounts are those given by Leroux‡ (1899) and Herrick§ (1902).

With regard to the actual frequency of arthritis as a complication the statistics collected in the present investigation show that it is far from common. It is probable that many cases have been overlooked or lost sight of in the older statistics, such as those of Vogelius from the clinics of Berlin, Paris and Munich, who found not more than 6 cases among 5158 cases of pneumonia, and those of Netter, who reported the same number among 4156 cases in German clinics, only a little over 1 per thousand.

Cave‖ was the first in this country (1901), to record an undoubted case of the disease and since then other cases have been reported here by Raw,¶ Dudgeon and Branson,** Secretan and Wrangham,†† and Nitch,‡‡ and in America by Howard,§§ Slaughter,‖‖ and Witt.¶¶ Still it is

* 'Clin. Soc. Trans.,' vol. xxxvii. 1904, p. 91.
† 'Bristol Med.-Chir. Journ.,' June, 1907.
‡ 'Brit. Med. Journ.,' 1901, vol. ii, p. 766.
§ 'Arch. Gén. de Méd.,' September 4th, 1906.

* 'Traité de la Pneumonie.'
† 'Wien. klin. Woch.,' 1888, Nos. 28-32.
‡ 'Les Arthrites à Pneumocoques,' Paris, 1899.
§ 'Amer. Journ. Med. Sci.,' vol. cxxiv, 1902, p. 12.
‖ 'Lancet,' vol. i, 1901, p. 82.
¶ 'Brit. Med. Journ.,' vol. ii, 1901, p. 1803, and vol. ii, 1906, p. 1400.
** 'Lancet,' vol. ii, 1903.
†† 'Brit. Med. Journ.,' vol. i, 1906, p. 916.
‡‡ 'Brit. Med. Journ.,' vol. ii, 1907, p. 720.
§§ 'Johns Hopkins Hosp. Bull.,' 1903, p. 303.
‖‖ 'Amer. Med.,' 1903, vol. v, p. 605.
¶¶ 'New York Med. Journ.,' vol. lxxxi, 1905, p. 25.

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probable that many cases of arthritis which are really pneumococcic are not recognised as such.

Pneumococcic arthritis usually appears as a complication after the crisis. One or more joints become hot, painful, swollen and tender. The larger joints are those more commonly affected, especially the knees, the hips and shoulders, but any joint may be affected. Redness and œdema are rare. There is some elevation of temperature but little constitutional disturbance unless other complications are present.

Affection of the joints results from a blood infection, and is in most cases accompanied by grave constitutional symptoms. Where the latter are present the prognosis is extremely grave. In some cases, however, the joint affection appears without toxæmic symptoms, and then the outlook is more hopeful. Out of thirty-one cases of pneumococcic arthritis in children recorded by Nitch,* fourteen were fatal, or 45 per cent. Out of sixty-three cases including all ages which I have collected from literature forty-one died, or sixty-five per cent. Periarticular inflammations and abscesses may also be met with. Arthritis is met with at all ages, but a considerable number have been recorded in quite young children. It may occur without being preceded by pneumonia, but following some other form of pneumococcus infection such as otitis media. The fluid in the joints in different cases may be thin, turbid or seropurulent, or purulent and of a thick creamy consistency. The treatment should be surgical by incision and drainage.

In view of the fact that the pneumococcus is such a frequent denizen of the secretions of the mouth and upper air-passages, it is not surprising that it should sometimes infect the mucous membranes, resulting in a variety of inflammatory conditions, erythematous, follicular, herpetic and and suppurative. Cary and Lyon† have reported a remarkable case of pseudo-membranous inflammation of the mucous membranes caused by the pneumococcus, in which nearly all the mucous surfaces of the body were affected with a profuse pseudo-membranous exudation. The mucous membrane of the lips, gums, cheeks, under surfaces of tongue, hard and soft palate, fauces, tonsils, pharynx, nose, the glans penis and the arms were covered with a membranous exudate consisting of thick white gelatinous material containing diplococci in almost pure cultures. There was also conjunctivitis with a flabby exudation from which the same pure culture was obtained. The passage of membranous shreds and mucus in the stools suggested that the gastro-intestinal tract was also affected.

The occurrence of croupous colitis as a complication of pneumonia was first recorded by Bristowe* in 1879. Osler† recorded cases where thin layers of adherent lymph were observed on the surface of the mucous membrane of large intestine, and one case in which there was a thick adherent exudation on the mucous membrane of the stomach. Since then a number of cases of pneumococcus infection of mucous membranes have been reported affecting mouth, tonsils, fauces. pharynx, trachea, nares, conjunctivæ, and rectum. Such cases may and do occur independently of pneumonia. Thrombosis was observed in twenty cases out of a total of 3066, or once in every 150 cases. It is nearly always the femoral veins which are affected and the left more commonly than the right. Michell Clarke‡ has reported a case in which there was thrombosis of the veins of the left arm and another in which there was reason to believe that there was thrombosis of the cerebral vessels. The external jugular and axillary were blocked in a case reported by Ashton and Landis. Of forty-one cases collected by Steiner§ death occurred in nine and there was recovery in twenty-five, while in the remainder the issue was not recorded. Of the twenty cases mentioned above, four were fatal, six recovered, and in ten the issue was not recorded. Thrombosis sets in with pain and tenderness in the region of the affected vessel, followed by œdema if the vein be blocked, and there is a rise of temperature.

Acute tonsillitis with exudate, severe angina or tonsillar abscess, may result from a pneumococcus infection, and is perhaps more commonly the cause of these affections than is at present realised. But as Cary and Lyon have pointed out, unless the pneumococcus is found in pure culture it cannot be accepted as the certain cause of the affections.

* *Loc. cit.*

† 'Amer. Journ. Med. Sci.,' 1901, vol. cxxii, p. 298.

* 'Theory and Practice of Medicine.'

† 'Trans. Path. Soc. Philadelph.,' 1885, vol. xii, p. 188.

‡ 'Bristol Med.-Chir. Journ.,' June, 1907.

§ 'Johns Hopkins Hosp. Bull.,' June, 1902, p. 130.

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It is interesting to note that the pneumococcus has been found by Rochon* in pure culture in the contents of herpetic vesicles about the anus. It is possible that the eruption of labial herpes in pneumonia is a local infection with the pneumococcus and is of the nature of an auto-vaccination. It is reported that the appearance of herpes in pneumonia is a favourable sign, and I am inclined to agree with that view. The favourable issue may possibly be influenced by this auto-vaccination.

Subcutaneous abscesses and cellulitis have resulted from local infection with pneumonia at post-mortem examinations.

Conjunctivitis and corneal ulcer and hypopyon have long been observed as following pneumonia, especially in children, but I have personally never observed these complications.

Nephritis has been observed as a complication in 12 out of 750 cases at St. Thomas's. Of these 8 were fatal. Nephritis appears to be a more frequent complication in youth than during other periods of life. Between the ages of 7 and 19 the mortality of pneumonia is very small, only 2·1 per cent. among the St. Thomas's cases, and it is significant that of the fatal cases 6 out of 8 had some kind of kidney complication.

Although the various complications have been considered individually it must be remembered that often there are a number of these co-existent. The condition is one of pneumococcal bacteriæmia, and we meet with a combination of lesions such as ulcerative endocarditis, arthritis, empyema and meningitis. Such cases are at present as far as we know invariably fatal.

With regard to treatment by means of serum and vaccines I have no personal experience to place before you. In judging of the effect of such treatment it is important to bear in mind the varying mortality of the disease at different ages. The great majority of young subjects recover under the usual methods of treatment ust as a large proportion of elderly subjects die. I must leave it to others to bring forward conclusive evidence that serums and vaccines are efficacious in saving life, especially at those ages at which it is most in peril.

As an encouraging indication that vaccine treatment of acute pneumococcic infections is promising and is deserving of careful and extended trial I may very briefly refer to a recent report by Dr. O. Boellke* on cases treated by him at the Städtisches Krankenhaus at Rixdorf. Fifteen cases in all were treated of which 13 were cases of pneumonia, all of a severe type, 1 was a case of empyema, and 1 was a case of pneumococcic endocarditis of malignant type. The cases of pneumonia all recovered except 2 which did not come under treatment until the seventh and eighth days of the disease when the condition was practically hopeless. Of the 11 cases which recovered 2 had one injection, 7 had two injections, and 2 had three, the average dose being ·5 c.cm., containing approximately 150 million pneumococci. The opsonic index was in all cases taken before the injections and again twenty hours after. As a rule the opsonic index was below normal before, and when this was the case it was invariably raised to normal or above normal after. In those cases where the index was found to be above normal before treatment it was either further raised or remained at the same level after. The most striking result, however, was obtained in the case of pneumococcic endocarditis. The opsonic index at first was as low as ·01. The first injection caused it to rise to ·53. Five injections in all were given during a period of twenty days. After fifteen days the temperature assumed a normal level and the patient made a good recovery.

Mr. A. G. R. Foulerton said that in a consideration of the possibilities of specific bacterial treatment of pneumococcic infection it was necessary, in the present state of our knowledge, to sharply distinguish between early stages of acute lobar pneumonia, when few would be inclined to use vaccine treatment, and the less immediately acute manifestations of pneumococcic infection, whether consecutive to pneumonia or not. In pneumococcal endocarditis, vaccine treatment apparently held out a very good prospect of success, and he had recently had a severe case in which the treatment seemed to give excellent results. But he assumed that the main object of the discussion was the treatment of acute lobar pneumonia. In considering the specific treatment it was necessary to differentiate between cases. It

* 'Journ. de Clin. et de Thérap. Infant,' Paris, 1896, p. 843.

* 'Deutsch. med. Woch.' September 12th, 1907, p. 1487.

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was necessary to regard first of all the earlier stages of every case of acute pneumococcal pneumonia, with a pure infection, and then cases in which the stage of resolution was prolonged and in which he thought there was often a secondary streptococcic infection. The causation of pleural empyema threw some light on the frequency of secondary infection. Dr. Mackenzie had said that nine cases out of ten were pneumococcal, but that did not accord with his own experience. Six years ago he had carried out a fairly careful examination of a series of cases of empyema, and found not only that the proportion of pneumococcic cases was lower than had been hitherto stated, but there was a considerable difference in the causation of the empyema as between adults and young children. In the case of adults, excluding the small minority due to other organisms, he found that 75 per cent. of the cases were streptococcic, and only 25 per cent. pneumococcic. In young children, however, excluding the negligible number of cases caused by other organisms, the percentage was more than reversed. Over 80 per cent. were pneumococcal, and less than 20 per cent. streptococcal. One had also to remember the characters of the sputum on microscopical examination in cases of pneumonia in which the stage of resolution was prolonged. Those who had examined a number of cases would know that streptococcic secondary infections were not uncommon. Therefore he suggested that at all events in some of the cases in older subjects in whom resolution was prolonged, treatment by streptococcic vaccine was worth trying. Similar treatment had been tried in at least one case of troublesome sinus discharge of streptococcic pus which remained after surgical treatment of the empyema. With regard to the treatment of the earlier stages of pure pneumococcic pneumonia there were two possible methods: (1) bacterial vaccination, and (2) serum treatment. The physician had to decide whether he would attempt to hurry on the process of auto-immunisation by injecting dead pneumococci into the patient, or whether he would supply the patient with ready-made anti-bacterial bodies in the form of a serum. He thought most would hesitate to advise treatment by vaccine within the first three or four days. Ehrlich, in his early work, pointed out that in the early days of infection there was generally a decrease of what might be termed the natural anti-bacterial substances in the blood, preceding the formation of the more intimately specific anti-bacterial bodies. There was a stage of lowered resistance to infection before immunity was acquired, now termed the "negative phase." So it seemed, on theoretical grounds, dangerous to give a patient in that stage of lowered resistance an extra dose of bacterial poison. Therefore, other things being equal, the serum treatment appeared to hold out the better prospect of success.

It must be acknowledged that the serum treatment had been up to the present a failure. But from the first some more or less satisfactory results had been obtained. Still the general use of different serums had not, he thought, been a success. The treatment was obviously uncertain in its results. During the last two years he and his colleagues had done a considerable amount of work at the Middlesex Hospital in that direction, on the advice of Sir Richard Powell, but their results had not been better than those of other people. Still, they appeared to have learnt wherein the failure lay in the treatment by serum. They commenced with two horses, one to be immunised against different strains of pneumococci, and the other against pneumococci and streptococci. After carrying out experiments for eight months both horses were unfortunately lost from other causes. But they had obtained a serum which protected rabbits in a certain degree. They then had a third horse, and after putting it through the course of immunisation for eight months they treated a number of cases. Seven of those cases might be taken as test cases, but the results in them had not been encouraging. In two of them he thought it could be said the serum had had a good effect. One was a very severe pneumonia treated on the third day, and the effect was as marked as it would have been in the treatment of diphtheria by a serum. In the other case, temporary improvement followed each dose of the serum, but the final result was unsatisfactory. There was nothing to be said about the other five cases, as they ran their normal course. He thought the cause of failure was, that whilst the horses' serum readily responded to pneumococcic immunisation with the production of anti-bodies, those anti-bodies disappeared rapidly from the horses' blood. After the serum had been used and the

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horses kept under treatment for nine months, all the immunising injections were dropped for six weeks, and at the end of that time one found the horses' serum had lost all evidence of the presence of anti-bacterial bodies, it failed to agglutinate the pneumococcus, the opsonic index was low, namely ·55, as compared with that of normal horses, and its protective effect on rabbits was lost. Then a large dose of pneumococcic vaccine was given, and within three days the blood showed very definitive agglutinative properties; it would agglutinate a culture of pneumococcus with a dilution of 1 in 25, and the opsonic index was raised to ·75. At the end of a week another injection was given, and the agglutinative power had risen rapidly. There was a good reaction, with a dilution of 1 in 500; an immediate reaction occurred visible to the naked eye, in the form of large clumps, with a dilution of 1 in 100. The opsonic index had risen to 1·5, and the protective effect on the rabbit was again manifest. The horse was then left alone for five weeks, at the end of which time all those properties had again disappeared from the blood. On consideration, the result seemed what one would expect from the knowledge on the pathology of human infection. In pneumonia, the anti-bodies produced by auto-immunisation did not seem to last, and recurrences were probably more common in that than in any other infective disease. Two years ago, Macdonald found that in a typical case of pneumonia the opsonic index rose about the day of crisis, and then fell to or below normal a short time afterwards. A similar loss of immunity was found experimentally in some rabbits. One rabbit had 4 c.c. of serum, and was protected by that against a very heavy dose of virulent blood from the heart of another infected rabbit. It was ill a short time and then recovered. One would have expected after that to have had a fairly lasting immunisation, but after re-inoculating that rabbit it died in the same time as one of the control animals. In the preparation of an anti-pneumococcic serum, one had to avoid over-immunisation on the one hand, and on the other one, had apparently to guard against loss of efficiency by the rapid disappearance of immune bodies from the blood. That difficulty occurred with all serums, but particularly with pneumococcic. Therefore he thought the failure, up to the present, was not due to any fallacy in the principle on which the treatment was founded, but to a want of knowledge as to the best method of producing the serum.

Dr. W. P. HERRINGHAM gave an explanation of the statistics on the matter which had been furnished from St. Bartholomew's Hospital by the Registrar, Dr. Langdon Brown. One table gave the mortality from the disease, and the other the frequency of the complications. The two tables were not drawn from quite the same series of cases. It was easy to give the death-rate from pneumonia, for it simply meant taking the cases in which pneumonia was given as the primary cause of death on the certificate. But in regard to the complications, Dr. Langdon Brown felt—and he agreed with him in that respect—that he ought not to omit from his statistics any case in which the physician during the life of the patient, or the pathologist after death, had put down pneumonia in the headings of the case. Therefore it included many cases in which pneumonia was the terminal infection of perhaps a long disease. For instance, one of the cases of gastric cancer was found after death to have grey hepatisation of the left lobe of the lung, and a portion of it was gangrenous. And many cases of chronic heart disease died in that way. Probably many of the cases were pneumococcal in origin. Still, if a person writing on pneumonia were to refer to the statistics as supplied, and say that was the percentage of complications, he would probably be misled. The table not only included those cases in which pneumonia, for the most part apyrexial, occurred as a terminal infection in a chronic disease, but also included cases in which the original disease was a general pneumococcic infection, and in which acute lobar pneumonia would occur not as the substantive or primary disease, but as a comparative accident in the course of general pneumococcic infection. Therefore in both ways the table of complications supplied required revision if applied to illustrate acute lobar pneumonia as the substantive disease, and if necessary Dr. Brown would be willing to alter it in that sense.

With regard to the sequelæ of pneumonia as a substantive disease, he first referred to local complications in the lungs. He would not refer to pleurisy and empyema. In the statistics there were four cases of gangrene and four cases of

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abscess of lung. But none of those cases of abscess could be truly called such; none of them were encapsuled: they were cases of sloughing areas of solid lung due to the virulence of the infection without any line of delimitation between that and the rest of the pneumococcic lung. It was perhaps natural that a condition should be at one time called abscess and at another time gangrene.

With regard to the cases which were called chronic pneumonia, cirrhosis of the lung and bronchiectasis, he had been over several of them but could only find one case in which there was the slightest doubt that the bronchiectasis, the contraction and the cirrhosis was the preceding condition. With regard to the cases of heart disease, St. Bartholomew's had an enormous percentage, but that was due to the fact that some of the Bartholomew's cases were acute pneumonia supervening on those general pneumococcic infections which were attended with ordinary ulcerative endocarditis and septicæmia. One of the cases was not even pneumococcal. It occurred in the practice of Dr. Tooth. A man was admitted for gonorrhœal septic endocarditis; gonococci were found in the blood and afterwards the man had acute pneumonia. He had been astonished how very few cases found after death to have pericarditis were recognised during life. That made him suspect that pericarditis was a much more common sequel of pneumonia than was at present believed. If so, it would mean that it was not nearly so fatal a complication as was supposed. Six cases of meningitis were discovered on post-mortem examination, and only one of them gave any symptom during life other than a somewhat drowsy delirium, such as was commonly found in pneumonia. The case which gave other symptoms than that gave rigidity of the right arm, strabismus, and so on, obviously pointing to meningitis, or a similar affection within the skull. The other cases were simple instances of delirium. That raised the question as to whether all cases of pneumonic meningitis were fatal, and whether some of the cases of severe delirium in pneumonia which recovered were really cases of meningitis or encephalitis due to pneumococcic infection? He did not know that anyone had tried to settle that point; he wondered whether anybody had looked at a series of optic discs in the course of acute pneumonia. With regard to the complications of acute nephritis, having followed many of the cases given under that heading he had not found one in which the acute nephritis led to a condition which lasted after the discharge of the patient from the hospital. Several of the cases were noted as having albuminuria when they came in, with a history of having had some albuminuria at a previous date. Several of the cases had a fairly acute nephritis, with hæmaturia, blood casts and considerable albuminuria, but in all of them the condition had cleared up by the time the patient left the hospital. Of phlebitis there had been a few cases at the hospital of the usual type, occurring in the legs only. Another point was the supervention of pneumonia in the course of other acute diseases. There had been three cases in which pneumonia occurred during typhoid fever, and in two of them it was a perfectly definite acute pneumonia occurring during the stage when the fever of the typhoid had subsided. In the other case the signs of pneumonia on one side of the chest were very distinct, but the fever was masked by the fact that the typhoid was at that time at the height of its course. There were two cases in which rheumatism was added, and they both occurred in the pneumonia wards. Rheumatic pneumonia was alleged to occur, and recently he had the case of a boy who was admitted for rheumatism and severe heart disease who had an extraordinary attack in his right apex which he thought must be consolidation of the lung. It was not at all a typical pneumonia. The cases were as follows: A man, æt. 28 years, was admitted for rheumatic fever, multiple arthritis and purpura. The heart was natural, and the rheumatism cleared up under salicylate of soda. Next day there was pneumonia of one base, and next day the other base was involved also. The fever fell by lysis in sixteen days. Another patient was a woman, æt. 31 years, who had rheumatism, double mitral disease, and later thrombosis of the left leg. A fortnight afterwards the left jugular vein was involved. There was dulness, bronchial breathing at both her bases, and thrombosis of the other leg. Lastly there were two cases of lupus erythematosus in which pneumonia occurred as a subsequent infection. One case was under Dr. Ormerod. The patient was a woman, æt. 24 years, and after lupus erythematosus she had pneumonia, peritonitis, septicæmia and pericarditis, and died. The other case was in his own ward, and was treated by Dr. Drysdale. She was ill for many months with lupus erythematosus, from which she recovered with loss of hair, little scar pits over her hands, and looking a skeleton. She was sent to her home. Within two months from that time she was admitted under his own care, and this time she had pneumococcal peritonitis, for which she was operated on and did well. A fortnight afterwards she had pneumococcal pericardial effusion, with pneumonia, which he was called in to see in the surgeon's ward. The pericardium was opened and drained, but after a short rally the patient died.

(*To be continued.*)

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A LECTURE

ON

EMPYEMA IN CHILDREN.

Delivered at St. Bartholomew's Hospital.

By A. E. GARROD, M.D., F.R.C.P.,

Assistant Physician to the Hospital, and Physician to the Hospital for Sick Children, Great Ormond Street.

THE setting apart of the diseases of children as a subject of special study, to which special departments are allotted in general hospitals, and which has acquired an extensive literature of its own, may be justified on a variety of grounds. A few diseases are practically confined to children, whereas from some they are practically immune. Many symptoms when met with in a child suggest an entirely different set of possible causes from those to which they are usually due in adults; but perhaps the most important ground is that almost every malady from which the child suffers receives a stamp, and takes its shape according to the age of the patient.

If you go round the medical wards of a children's hospital you will find that at any time a majority of all the patients under treatment are victims of the invasion of three chief micro-organisms, the tubercle bacillus, the pneumococcus and the organism of rheumatism. Other bacteria contribute their share, such as the meningococcus, and those which are responsible for gastro-intestinal disturbances; and the exanthemata and other infectious fevers which are treated apart contribute, of course, a large share of the illnesses of childhood.

Concerning the peculiar features of rheumatism as seen in children much has been written, and has become a part of current knowledge. That the joints are much less subject to attack than in adults, that the heart is specially prone to suffer, and that certain manifestations of the disease, such as rheumatic nodules and chorea, are far commoner

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in children even than in young adults are facts known to all. What is true of rheumatism is also true of other diseases, for they likewise receive the stamp of childhood.

Thus certain tubercular manifestations are far commoner in children than in adults, as, for example, tubercular meningitis, peritonitis, and arthritis, which bulk largely among the tubercular cases in children's hospitals, but which are comparatively uncommon amongst adult patients. Again, if we turn our attention to that most important disease which results from the invasion of the pneumococcus, we shall find that here also the same holds good. Whereas in adults the common manifestation of the pneumococcal disease is the familiar lobar pneumonia, pneumonia in children is far more often of the bronchial form, and in children such results of pneumococcal infection as otitis media and empyema are much more frequently met with.

It is one of the chief difficulties by which we are met when we attempt to arrive at a satisfactory classification of diseases that the same morbid event may have a variety of pathological causes. Thus whilst, on the one hand, it would be preferable to treat of empyema as a part of that great disease which results from pneumococcal infection, it is necessary on the other hand to consider it from the clinical standpoint as a definite entity which has very different causations in different cases.

Whereas in children empyema is in the great majority of cases due to the pneumococcus, and occurs as a sequel to pneumonia, Dr. Samuel West states, in his work on 'Diseases of the Organs of Respiration,' that in adults it is most commonly produced by streptococcal infection. Other organisms are also concerned in its production in some cases, *e. g.* the tubercle bacillus and the staphylococci. Empyema may be met with as one of the manifestations of a general pyæmia, or it may result from perforation of the diaphragm by an abdominal abscess. Again, it may be developed in the course of an infectious fever, such as scarlatina.

In a case of the kind which is most commonly seen in children, in which an empyema develops as a sequel to an attack of broncho-pneumonia, the secondary fever due to the empyema may be continuous with that of the primary illness. More often, after the temperature has fallen and has remained normal for a few days, a secondary rise occurs; but the fever, which is usually of a remittent type, is not often so high as that of the preceding pneumonia; it may be very slight indeed, and the temperature during the development of the empyema may he scarcely raised above the normal. I take it that the use of such lectures as these is to underline and emphasise practical points which may be of use to you in your practice hereafter, and it is one of the special points which I desire to emphasise, that a slight degree, or even the absence of febrile disturbance in no way excludes the diagnosis of pus in the pleura.

It often happens that what arouses the suspicion of empyema is the appearance of the patient. Increasing pallor, an aspect of grave illness and progressive loss of strength, instead of the improvement which is to be expected in a patient who is recovering from an attack of pneumonia, suggest a diagnosis, which must, however, be based to a great extent upon physical signs. I say to a great extent because the physical signs may prove ambiguous or even deceptive. No one who has not had considerable experience in examining the chests of young children can realise to the full how misleading physical signs may be in such cases. The diagnosis by their means between consolidation of a lung and pleural effusion in a young child is often a matter of much difficulty, and not infrequently is impossible even to the highly-trained observer.

In not a few cases of empyema the physical signs allow of no doubt as to the presence of fluid in the pleura, but whether the fluid is serous or purulent cannot be diagnosed by their means, unless the abscess be actually pointing beneath the skin.

The entire side of the chest may be bulged and the intercostal spaces may protrude. There is a conspicuous sense of resistance over the dull area, vocal vibrations are abolished, the heart is obviously displaced and the liver may be depressed. The cases which present difficulty are those in which the empyema is a small one, and shut in by adhesions, and in which the diagnosis has to be made from an unresolved or slowly resolving pneumonic consolidation. Such consolidated lung may be covered by a thick layer of recent lymph, which is as effectual in damping vibrations as is an actual layer of fluid. Moreover, in the case of a young

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child it is often impossible to avail ourselves of those signs which are connected with the voice, and which are often of so much value in the detection of fluid in the pleura. Bronchial breathing is often well heard over the area of an effusion, whether serous or purulent, and on the other hand feebleness of breath-sounds may be due to a layer of lymph. If localised œdema of the chest wall were a more common sign than it is, it would prove a great help in the detection of empyema, but it is so uncommon as to lose much of its importance. In doubtful cases the prudent man will suspend his judgment until the exploring needle has been employed, for often it alone can give the required answer. The point which has to be decided is whether or no the signs are sufficiently suggestive of the presence of fluid to warrant exploration.

I need hardly point out to you that exploration of the chest is a proceeding which is not wholly devoid of risk. Puncture into a solid and adherent lung may lead to the development of most extensive surgical emphysema, which may extend far beyond the limits of the chest wall, and I have once seen a pneumothorax produced by a puncture; but far more harm is likely to result from omitting to explore when exploration is called for, than from a puncture or punctures which may prove to have been unnecessary, provided always that the trifling operation is carried out in a careful manner and with due aseptic precautions. Even the exploratory puncture may mislead. Around a localised empyema there may be a considerable area of consolidation of lung or a zone of inflamed and adherent pleura, and through such regions, instead of into the area of suppuration, the needle may pass. Again, an empyema which yields conspicuous physical signs may be enclosed between the lobes of the lung or between the base of the lung and the diaphragm, and even repeated punctures into the dull area may fail to reveal the presence of pus.

As a rule the collection of pus is situated towards the base of the lung, over its posterior or axillary aspect, but the presence of an empyema cannot be excluded on the ground that the physical signs are in an unusual situation. In the case of a child still in this hospital an illustration of this point was afforded. The empyema which followed an apical pneumonia lay over the apex of the lung on its posterior aspect, and the exploring needle which revealed the presence of pus was inserted high up in the interscapular region.

The pus removed varies widely in character in different cases. Often it is thick and creamy, but not uncommonly the fluid is only conspicuously turbid. Even serous fluid which proves sterile and is devoid of leucocytes may show a slight turbidity, but a microscopic examination of the sediment from a doubtful sample will usually serve to prove or disprove its purulent character.

In cases in which empyema results from perforation of the diaphragm by an abdominal abscess, the pus usually has an offensive odour, but even in cases in which there is no question of such an origin, offensive pus may be found. This was the case with the pus from the apical empyema which has already been referred to, and a bacterial examination revealed the presence of the Bacillus coli communis, as well as of the pneumococcus, which was doubtless the organism to which the suppuration was primarily due.

Among the means of diagnosis available in cases of empyema examination by the Röntgen rays must be referred to. The area of the effusion appears deeply shaded, the line of the diaphragm may be indistinct, but the want of movement of the diaphragm may be established in some cases.

A leucocyte count often proves of much service especially in cases in which the physical signs are equivocal, and in which there is little elevation of temperature. A leucocytosis of 30,000 to 40,000 is common, and may turn the scale in favour of exploratory puncture.

There are some morbid conditions in which the arrival of an accurate diagnosis is not supremely important from the standpoint of the patient, because the treatment of the case will not be determined thereby in one direction or another. This is by no means the case with empyema, since the presence of pus in the pleura calls for prompt action. Appropriate surgical treatment will often save the life of the patient, and at the least may be expected to save him from a prolonged and severe illness.

If the lesion remains undiagnosed and untreated one of several events may happen. Some empyemata undergo spontaneous resolution, if the collection of pus be small and circumscribed. This is by far the most favourable natural ending, but the

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adjacent portion of lung is left bound down by tough adhesions. Spontaneous rupture may occur into the lung. This event was known to the ancients and was regarded as a favourable result which may lead to spontaneous cure. However, the evacuation is apt to be incomplete, and if the collection of pus be a large one, this event may prove fatal, the patient being in a sense drowned by the filling of the bronchi with pus. Contrary to what might be expected, even when the pus is percolating through the lung for some time, there is little risk of the setting up of pneumonia or of other damage to its structure. Nevertheless I would urge you not to allow the fact that the patient has begun to spit up his empyema to deter you from prompt operative interference, but rather to regard it as an additional incentive thereto.

Rupture into the pericardium or through the diaphragm is naturally a very grave event. Penetration to the surface, which often occurs in the neighbourhood of the nipple, is not uncommon in untreated cases, but the opening seldom allows of satisfactory drainage, and although spontaneous recovery may be so brought about, it more commonly happens that the patient succumbs in the end to prolonged suppuration with resultant lardaceous changes in various organs. The collapsed lung may be so bound down that it cannot expand, and in adults especially the chest walls may be unable to fall in so as to close the cavity, whilst the pleura may become increasingly thickened and infiltrated with calcareous deposit. I can recall one instance, in an adult, in which it was necessary to employ a hammer and chisel to make a counter opening through the pleura.

The complications of empyema consist chiefly in the extension of inflammation to the adjacent parts and the formation of metastatic abscesses. In pyæmic cases the empyema may itself be regarded as a metastatic abscess, but secondary collections of pus may form in connection with primary empyemata.

When a neighbouring serous cavity, usually the pericardium, is invaded the affection is not necessarily purulent. In cases in which the collection of pus is adjacent to the pericardium, a well marked pericardial friction-sound may develop and may disappear in time, without any evidence of pericardial effusion being forthcoming. Purulent pericarditis, which is one of the gravest of the complications of empyema in children, does not usually manifest itself by any unequivocal physical signs. The diagnosis, if made at all, is usually based upon the general condition of the patient. When suspected pyopericardium may not be present, and when present it is often not suspected during life. The difficulty or even impossibility of recognising purulent pericarditis in not a few cases is a deplorable fact to which I wish to draw special attention, for if the pus in the pericardium could be recognised, it is probable that some lives at least might be saved. In connection with purulent pericarditis no friction-sound is usually heard. Enlargement of the area of cardiac dulness may be misleading. In a case which was recently under my care at the Hospital for Sick Children an empyema was opened on the left side. The child did not make satisfactory progress; the temperature continued to be raised. On general grounds, purulent pericarditis was suspected, and a bulging swelling in the region of the pericardium could be felt through the operation wound. In this case the Röntgen rays proved of the greatest help; a great extension of the cardiac shadow was revealed, and acting upon these indications my colleague, Mr. Steward, opened the pericardium by means of blunt forceps, through the wound of the previous operation, and evacuated four ounces of pus therefrom. The patient has made a complete and satisfactory recovery.

The results of drainage of the pericardium in cases of purulent pericarditis are by no means always so favourable, and, as Dr. Samuel West has pointed out, the outcome depends to some extent, upon whether the purulent pericarditis is merely a complication of an adjacent empyema or is one of the manifestations of a general pyæmia. In the case referred to a bacterial examination showed that the pericarditis, like the original empyema, was due to a pneumococcal infection.

The short remainder of time at my disposal must be devoted to the treatment of empyema. Here the general rule must be that when pus has been found to be present it must be evacuated. The concensus of opinion among those who have much experience of such cases in children favours operation without delay, including the removal of a portion of a rib.

It may be that the general condition of the patient may lead to the postponement of the operation for a day or two, and, when the collection of pus is so great and the displacement of viscera so conspicuous that urgent distress is caused, it may be thought best to remove a portion of the effusion by aspiration, before completely evacuating it. Undoubtedly some empyemata have been successfully treated by aspiration alone, but it is not to be recommended as a routine treatment although it may be desirable as a temporary measure in some cases in order to afford partial

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relief before operation, or when for any reason an operation is for the moment contra-indicated.

It rarely happens that there is more than one pus-containing cavity in the pleura, a condition which is known as loculated empyema. In such cases the first operation may not suffice for the evacuation, and a second opening, either from the original wound or in another portion of the chest-wall, may have to be made.

Double empyema is not common, and is usually met with in very young children. When confronted with it you may have great searchings of heart as to what is the course which you should pursue. Experience shows that the difficulty of dealing with the condition is less than might be supposed. In opening a small empyema the general pleural cavity is seldom laid open, and the collection of pus is usually circumscribed by adhesions. With due caution it will usually be possible to deal with the two sides one after the other, but it may be necessary to resort to aspiration on one side as a temporary measure. The course to be followed in any particular case must be determined by the special circumstances, but here, as in single cases, the rule holds good that when pus is found it should be evacuated as soon as this can be done compatibly with safety.

January 6th, 1908.

THE PRACTICAL VALUE OF BLOOD-PRESSURE ESTIMATION.

A paper read before the South-Eastern Branch of the British Medical Association.

By LEONARD WILLIAMS, M.D., M.R.C.P.,
Physician to the French Hospital; Assistant Physician to the Metropolitan Hospital.

I PROPOSE in this paper to call your attention to the practical value in every-day work of a comparatively new aid to diagnosis which is deserving of a much more general recognition than it has so far received, namely, the estimation of degrees of blood-pressure. I have nothing new or startling to say on the subject, my intention being merely to emphasise certain facts and considerations which, though known, seem to be insufficiently utilised.

There are many means by which blood-pressure may be estimated. Of these, some are good, others indifferent, and one, at least, is altogether bad. Of the good ones, through the kindness of Mr. Hawksley, I bring two specimens to show you; the one a mercurial instrument,* the other an alcoholic,† between which in the matter of accuracy there is substantially nothing to choose, but in the matters of portability and durability the latter is to be preferred. Another good method is that of measuring what is called the stability of the pulse. In the normal individual the pulse-rate, standing, is six to ten beats per minute faster than the pulse-rate recumbent. If this difference becomes markedly augmented, it means that the tone of the peripheral arteries is deficient; that there is, in fact, undue vaso-dilation and low blood-pressure. If the difference is abolished, it means that the tone of the peripheral arteries is excessive; that there is undue vaso-constriction and high blood-pressure. If the difference is reversed, so that the rate standing is slower than the rate recumbent, it means that there is definite organic disease in the arteries over a considerable area, with permanently high blood-pressure. This very simple method is of very great importance, more especially, as will presently appear, where the estimation of subnormal blood-pressures are in question.

Of the indifferent methods, I also bring a specimen to show you. It acts by compressing the radial artery against the wrist by means of an air-cushion. It has the superficial attraction of fitting into the waistcoat pocket, which does not, unfortunately, counterbalance the grave disadvantage of utter unreliability. All the instruments which depend upon this compression of a single artery against a bone—and there are several—are equally unsatisfactory and untrustworthy.

The method which is altogether bad is that which, I am sorry to say, is most generally employed, namely, the palpation of the artery at the wrist. When I see a man adopt this method and thereupon proceed to pronounce a positive opinion my heart goes out in sympathy to him, because if he is an honest observer and not a mere poser, I know that a day of bitter disillusion awaits him. The reason of this is that our tactile sense is capable of estimating total force only; it is, however well educated, quite incapable of estimating total force per unit of surface, which is absolutely essential to the end in view. Of this method, then, it may be said, to borrow Lord Beaconsfield's

* Stanton's modification of Riva Rocci.

† Oliver's sphygmomanometer.

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famous words in connection with a certain policy, that "it is not only dead but damned."

To recapitulate; if we wish to arrive at any accurate estimate of the blood-pressure we must use an instrument with a 12 c.mm. armlet; either one of the modifications of Riva Rocci's mercurial manometer or Oliver's alcoholic manometer; but if, in the absence of such an instrument, we wish to gain information as to the state of the elasticity of the peripheral arteries, the factor upon which we know the physiological adjustment of blood-pressures mainly to depend, then we may do so by counting the pulse-rate in the upright and recumbent postures. Outside these means, all is vanity and vexation of spirit.

The normal blood-pressure is about 120–130 mm. Hg. It is lower in women, and still lower in children. It increases with advancing age. It is temporarily, but considerably, raised during muscular exercise, by mental excitement, by stimulation of a sensory nerve, and to a less extent by various physiological processes, chief among which are menstruation and digestion. It is normally lowered after muscular exercise, by fatigue, whether mental or muscular, and by fasting. The pressure is augmented by the application of external cold, and is lessened by the application of external heat. In short, whatever constricts the arterioles increases the pressure, whatever dilates them reduces the pressure. Now, there are certain agents, both endogenous and exogenous, which play the part, some of them of vaso-constrictors, others of vaso-dilators. Among drugs we know that ergot, opium, digitalis, and strychnine are all vaso-constrictors, and that nitrite of amyl, nitro-glycerine, erythrol tetranitrate, aconite, and chloral are vaso-dilators. Similarly, we know that certain of the internal secretions, such as that of the supra-renal, are strongly vaso-constrictor, while others, such as the ovarian, are believed, in their nascent states at any rate, to be definitely vaso-dilator. And so it is with the poisons of certain diseases. Some of them tend to constrict the arteries, others to dilate them.

Let us from this point of view consider the two most important chronic diseases which afflict people in this country, namely, goutiness and tuberculosis. The poison of gout, whatever it may be (and it is certainly not uric acid) is a powerful vaso-constrictor; the toxines of the bacillus of tubercle act strongly as vaso-dilators. Speaking generally, one would say that the differentiation between these two conditions is already so well defined that to insist upon any additional points of dissemblance is merely to paint the lily. On further consideration, however, it is obvious that both of them being protean and ubiquitous, there may be considerable resemblances between their manifestations in certain parts of the body. I have known dermatologists sorely put to it to decide which of the poisons was responsible for the production of certain lesions, and we can probably all of us remember cases in which a subacute arthritis has been positively assigned to the one cause when it ultimately proved to be due to the other.

The estimation of the blood-pressure in such cases will often throw a flood of much-needed light upon the question of diagnosis, for the gouty cases will always have a well sustained blood-pressure, whereas the tuberculous will always tend to be below the normal. And here let me say that the manometer will never tell us as much as we want to know about subnormal blood-pressures. On the subject of those which are excessive, it speaks eloquently, decisively, and with an authority from which there is no appeal. But blood-pressure is never unduly low to the same degree or in the same sense that it is sometimes unduly high. The reason of this is that if the pressure falls below a certain level, which has been estimated at about 60 mm. Hg., the stream becomes so sluggish that the blood does not reach the medulla in sufficient quantities to keep the animal alive. When possible, therefore, the medulla will always see to it that the pressure is kept at or about the normal level. When the heart is at fault, by ordering a vaso-constriction, it sends the arteries to the rescue; when the arteries are at fault, by increasing the number of systoles, it sends the heart to the rescue. Thus it is that when we suspect the blood-pressure to be tending to the low side, it is better to disregard the manometer and seek our information by interrogating the stability of the pulse. Consistently low blood-pressure, such as that which obtains in tubercle, is caused by want of tone in the peripheral vessels, and this is exposed more effectively by a wide divergence in the pulse-rates than by the readings on the manometer.

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Dyspnœa is a symptom both of goutiness and of tubercle. It is no doubt a symptom of various other conditions, but in these latter it constitutes merely one detail in a picture which, when regarded as a whole, is readily recognisable. The dyspnœa to which I now refer is on a different level. It is slight, but persistent and slowly progressive. It is not obtrusive when the patient is quiescent, but it is liable to become so upon the least effort. An examination of the heart and lungs reveals nothing at all definite. The heart is not enlarged and there are no murmurs; the chest moves well and the respiratory sounds are normal. There is no history of a recent illness, the patient is not losing flesh: he appears, on the contrary, to be in good health except for this breathlessness. In explanation of his condition he employs one of the numerous, apparently satisfying, but utterly meaningless formulæ such as "run down" or "below par," which our grandfathers invented to cloak their ignorance, and which now rise up to confuse us. But such an explanation we know to be insufficient, and we remind ourselves of all we know concerning unattached dyspnœas. The only two which appear to us to have any bearing on the present case are those which occur in gouty subjects and those which forerun, often by a considerable lapse of time, the explosion of some definite tuberculous manifestation, and we narrow the case down to one of these two. We obtain no assistance from outside considerations such as age, occupation, family history, and the like. The case must be judged on its present merits, and we examine it again with this end in view. In such an investigation, to deprive ourselves of the information afforded by the estimation of the blood-pressure is, Nelson-like, to put the telescope to the blind eye. For if the dyspnœa be accompanied by a high blood-pressure, a manometer reading of, say, 180 mm. Hg., then our clinical picture is practically complete. We know that we are in the presence of functional high arterial tension due to the constriction of the peripheral vessels by the action of toxines of gastro-intestinal origin, and that if we would cure the dyspnœa and forestall the supervention of definite arterial degeneration, whether it be in the aorta, in the kidneys, the liver, or elsewhere, we must not only check the supply of these toxines by a severe restriction of the intake of animal foods, but we must also ensure the elimination of those already present, by mercurial purgation, by sudorifics, and, more especially, by calling upon the kidneys, through the agency of theobromine, to increase their excretory activities. We shall, further, be well advised if, with a view of ferretting out such of the toxines as seek to lurk in the lymph spaces, we administer iodide of potassium in fairly large doses; but let me warn you against giving such drugs as nitro-glycerine and erythrol tetranitrate, which are vaso-dilators pure and simple. These are excellent servants in an emergency, but inasmuch as they attack a symptom without in any degree contributing to the removal of the cause, their employment in chronic cases is as unscientific as it is liable to be disastrous.

If, on the other hand, the dyspnœa which we are endeavouring to track to its lair should turn out to be accompanied by a low blood-pressure, we can afford to exclude any causal connection with the gouty diathesis, and our attention is immediately turned in the direction of tubercle. The dyspnœa of tuberculosis is liable to appear very early in the disease, so early in fact that it may easily be the only symptom present. When, therefore, it is associated with the vaso-dilation which we recognise as one of the physiological effects of the tuberculous toxine, we are in the presence of a combination which, although by no means pathogenic, is so suggestive as to arouse suspicions which compel the most careful investigation.

The value of estimating the blood-pressure in a diagnostic difficulty of this kind is evident, for the treatment of a gouty dyspnœa differs so widely and so fundamentally from that which is proper to a dyspnœa of tuberculous origin that anything which will save us from an error in this respect is surely to be welcomed with open arms. Nor is dyspnœa the only symptom about which we may find ourselves in a difficulty in this connection. Bleeding from the nose is very often tuberculous and accompanied by low pressure; it is no doubt more often due to high arterial tension, but it is very desirable that we should employ such means as are open to us to decide between the two causes. The same may be said of hæmoptysis. This is commonly due either to tubercle or to mitral stenosis, but it is often, I may say very often, caused by the rupture of a branch of the bronchial arteries of the systemic system, and is the direct

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result of high arterial tension. If we can discount the "hypertensive" effects of anxiety and excitement at the sight of blood, the manometer will always enable us to distinguish such a hæmoptysis from one due to tubercle, though it will not help us to decide between a hæmoptysis due to mitral stenosis and one due to high arterial tension. The reason for this is that the arterial blood-pressure in mitral stenosis is always high, because the venous pressure is always high, and the physics of life demand that the arterial pressure shall always be definitely higher than the venous.

Another symptom which may point in two diametrically opposite directions, in the decision between which we may obtain invaluable help from the study of the blood-pressure, is albuminuria. Albuminuria is a symptom whose real significance is not generally appreciated at its true value, for its importance is usually grossly exaggerated. It is, of course, an accompaniment of very grave and fatal diseases, but it is also present in disorders which, when properly treated, are very trifling and transient. Albumen appears in the urine whenever the kidneys are passively congested, and it requires no great effort of the imagination to realise that such a state of matters may occur in people who are structurally perfectly sound. Collier, of Oxford, has shown that young men in training, who may consequently be considered to be in the pink of physical condition, develop a transient but abundant albuminuria after hard exercise. The explanation of this is that the vaso-constriction which takes place during muscular action is followed by a reaction in which the vessels are unduly dilated, and that this vaso-dilation in the splanchnic area, "the splanchnic lake" or "abdominal pool" as it has been called, favours a passive congestion of the kidneys which shows itself by the presence of albumen in the urine.

The albuminuria of adolescents, which is called "cyclical," "postural," "physiological" or what not, is due substantially to the same train of events. In these cases, so long as the patient is recumbent, no albumen appears in the urine. No sooner, however, is he placed in the erect posture than the albumen appears. Now, the normal arterial response to change of posture is a vaso-constriction when the person rises from the recumbent to the upright position. If this response is inadequate, there immediately ensues a passive filling of the vessels in the splanchnic area with consequent albuminuria. In adolescents, especially in those who, as the saying is, have "outgrown their strength," this response is peculiarly liable to be inadequate, as it is in convalescents from acute disease, or those who from any cause, such as a fractured thigh, have been obliged to keep their beds for any length of time. We have already seen that the toxine of tubercle is vaso-dilative in its effects, and we know therefore that a passive congestion of the renal area with consequent albuminuria is to be expected in such cases. It would be possible to multiply instances, but what I wish to insist upon is that the simple expedient of estimating the blood-pressure both by the manometer and the postural method will generally enable us to place an albuminuria in its proper category. The conditions in which albuminuria is due to actual disease in the kidneys are usually accompanied by high pressure. In chronic interstitial nephritis this is so notoriously the case that granular kidney was until recently, and is still far too frequently, regarded as the sole cause of supernormal pressure. In the other forms of nephritis the pressure varies, but it is generally high, and is seldom or never as definitely subnormal as in the cases we have just considered. This, at any rate, it is safe to say, that where there is albumen in the urine it is wrong to make a diagnosis of kidney disease unless the view can be sustained by the presence of casts or some equally conclusive confirmatory fact; and he who allows himself to be alarmed by the presence of albumen into prognosticating evil for the patient, without availing himself of the assistance to be obtained from estimating the blood-pressure, does a very unwise thing for himself and one which is liable to be very harmful to his patient. For, consider for a moment the differences in treatment which the two categories necessarily impose. In the case of the diseased kidney it is essential that nitrogenous waste should be reduced to a minimum. Accordingly meat foods are removed from the dietary. It is further necessary to call upon the other emunctories to assist in the work of excretion, by administering purgatives, hot baths and other sudorifics. The life of the nephritic must be restricted; his responsibilities must be reduced, and he should if possible be sent to a

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warm, equable, relaxing climate. Now, if a person who is suffering from mere vaso-dilation is treated on these lines, not only does he fail to improve, but he becomes rapidly worse, both subjectively and objectively. Such a person requires above all things a stimulating *régime*, he demands cold baths instead of hot, strychnine, ergot and other vaso-constrictors, the salts of calcium (especially the chloride) and other cardiac tonics, a bracing climate and not a sedative. Treated in the wrong way he becomes a chronic invalid, a hypochondriac, a nuisance to himself and everyone connected with him. Treated in the right way he recovers rapidly and completely, and is fitted to take his proper place as a useful member of society. If the information afforded by the manometer can make all this difference in the fate of but a single individual in a single year, it is surely an instrument which is well worthy of respect and commendation.

In connection with dyspnœa I have spoken of functional high arterial tension, the state, that is, of general vaso-constriction which is brought about mainly by the absorption of toxins from the gastro-intestinal canal. The symptoms of this condition are very indefinite. They consist, sometimes, though not always, of dyspnœa, of malaise, of disinclination for exertion, bodily and mental, slight constipation, slight polyuria, nervousness, irritability, somnolence, inability to concentrate attention, distaste for everything that used formerly to interest. Now such is substantially the description of the condition to which the term neurasthenia is applied, and the diagnosis between the two is very liable to provide a pitfall for the unwary. Here, again, the estimation of the blood-pressure will prevent a mistake which is liable to be fraught with the most serious consequences. The term "neurasthenia" is a very convenient peg on which to hang loose diagnosis; it is classical, sonorous and satisfying, and I am afraid that when in difficulties we all resort to it more frequently than we should. There are some cases in which we may do this with perfect impunity, but there are others in which we do so at our own and our patient's peril. And the key to the situation is the estimation of the blood-pressure. In true neurasthenia, in real exhaustion of the nervous centres from whatever cause arising, the blood-pressure is low, lower than it is in any other chronic malady, except the terminal phases of cancer, tubercle and other wasting disease. In the other, the gouty, the gastro-intestinal condition, the blood-pressure is very high; such is the essence of the complaint, so that the manometer will always inform us in quite unmistakable terms as to which of the two we are dealing with. The importance of reaching a right conclusion on this point resides, as in the case of albuminuria, in the differences in the treatment proper to the two conditions. In neurasthenia, rest and overfeeding is the orthodox line of treatment, and a very successful one it usually is; but if we adopt such a policy in the condition in which high tension is at the root of the mischief, we shall certainly increase this tension to the point, perhaps, of causing the rupture of an important artery and inducing a devastating and crippling hæmorrhage. The same considerations apply to the management of post-influenzal conditions. These are usually characterised by nervous exhaustion, and up to a certain point are properly treated by a tonic *régime*. It is, however, all too easy, especially in those past middle life, to push such a *régime* too far, and this we are very likely to do if we fail to use some reliable means for measuring the blood-pressure. I have seen grievous and irreparable injury wrought in such cases by over-persistence in the use of strychnine and its congeners, injury which could never have occurred had the verdict of the manometer from time to time been consulted.

I have left myself no time in which to speak of the prognostic value of estimating the blood-pressure; how, for instance, the manometer may assist us in assigning a sudden collapse in typhoid either to hæmorrhage or perforation; how the outlook in apoplexy is influenced by the number of mm. of mercury which the instrument registers; or what information we may gain from the same means when called upon to decide between the various causes which may give rise to sudden unconsciousness. I trust, however, that I have said enough to support my plea that the manometer is not merely, as is sometimes wickedly suggested, a pretty toy with which the *dilettante* may suitably amuse himself, but that it is a true workman's instrument of immense practical utility, upon whose verdict the most momentous issues may often depend.

January 6th, 1908.

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PNEUMONIA AND ITS COMPLICATIONS.*

By HECTOR MACKENZIE, M.D., F.R.C.P.,
Physician to St. Thomas's Hospital and to the Brompton Hospital.

AND DISCUSSION THEREON BY

Mr. A. G. R. Foulerton.
Dr. W. P. Herringham.
Dr. W. Pasteur.
Dr. W. J. Hadley.
Dr. N. Dalton.
Dr. A. M. Gossage.
Dr. A. C. Latham.
Dr. H. A. Caley.
Dr. S. West.
Dr. N. I. C. Tirard.
Dr. J. Fawcett.
Dr. A. B. Harris.
And Dr. W. L. Brown.

(*Continued from p.* 192.)

Dr. W. Pasteur said it would be impossible, within the time allotted to each speaker, for him to deal fully with all the data furnished him by the kindness of Dr. Williamson, their medical registrar, and of his house physician Mr. Apperly. He would therefore content himself with a brief reference to the pneumonia statistics of the Middlesex Hospital, and then deal somewhat more fully with the cases of acute dilatation of the stomach and of gangrene of the lung. Of 582 cases treated during the period under review, 142 died, giving a case-mortality of 24·3 per cent. The exclusion of cases under ten, the decade during which the mortality from lobar pneumonia was at its lowest, made it hazardous to compare those figures with other records, which generally comprised cases at all ages, but it might, he thought, be assumed that the figures presented then would show a somewhat higher general mortality rate than would have been the case had all ages been considered. The mortality for the second decade was 5·2 per cent., for the third 14·2 per cent., for the fourth 34·3 per cent., for the fifth 39·3 per cent., for the sixth 46·6 per cent, and over sixty 68·6 per cent. Complications were met with in 72 cases, 15·5 per cent. Of those 39 died and 33 recovered. Among the more frequent complications the most fatal were, pericarditis (13 deaths in 17 cases) and gangrene of the lung (5 deaths in 7 cases.) There were 28 cases of empyema of which 21 recovered. With three or four exceptions the signs of pericarditis (friction) were recognised during life. Effusion was generally slight, never large in amount. Acute dilatation of the stomach occurred twice, and was in each case the immediate precursor of death. The first case was that of a very stout married woman of 24, admitted to hospital on the second day of an acute attack of pneumonia of the right lower lobe. On the fourth day after admission there was frequent vomiting of brownish fluid, without effort. At that time the stomach was not obviously dilated. Twelve hours before death, which took place on the following day, dilatation of the stomach was recognised, giving rise to marked bulging behind and below the left ribs. It was accompanied by extreme dyspnœa and increasing cyanosis with irregularity of breathing and of heart action. At the post-mortem examination the U-shaped dilated stomach occupied nearly the whole of the abdominal cavity, reaching to the pubes. It contained 35 ounces of dark greenish fluid, and on being relieved of its contents rapidly shrank to little more than its normal size. The second case was that of a poorly nourished woman, æt. 36 years, admitted with pneumonia of the left lower lobe. She died within twenty-four hours of admission. Symptoms of acute dyspnœa and rapidly increasing cyanosis, attributed to heart failure, supervened four hours before death, with irregularity of pulse and respiration. Post-mortem examination revealed an enormous U-shaped dilatation of the stomach and first part of the duodenum. He had met with a third example of that complication in private with symptoms very similar to those just described. In that case the dilatation of the stomach was very obvious. In a series of forty-four cases of acute dilatation analysed by Campbell Thomson there was associated pneumonia in three cases, including the first case just referred to. They might therefore conclude that that complication was not of extreme rarity. The treatment of the condition had so far been very disappointing, even in cases not complicated by a serious constitutional disorder. The presence of pneumonia, in his opinion, negatived the resort to heroic measures, such as laparotomy for the relief of suspected pressure on the duodenum, or kinks at the pylorus. The best hope of success in such cases would seem to lie in an early recognition of the trouble, before the dilatation had proceeded far. The occurrence of vomiting or an unexpected aggravation of the dyspnœa should direct immediate attention to

* An address delivered before the Medical Section of the Royal Society of Medicine.

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the stomach, and on the least indication of dilatation its contents should be emptied by means of a soft tube, all fluids by the mouth withheld and rectal injections of normal saline solution or plain boiled water with brandy substituted. Failing a tube, the emptying of the stomach in cases with excessive secretion might be facilitated by rolling the patient into the prone position as in a case reported by Mayo Robson and Moynihan, while strychnine in full doses should be given hypodermically to counteract the tendency to collapse. There were among their cases seven instances of gangrene of the lung, six men and one woman. With two exceptions the patients were over forty. There was a clear history of alcoholism in four and a presumption of it in another. In two of the cases alcohol was not a factor. Only two of those patients recovered and one of them died a year after from purulent meningitis following on bronchiectasis. As the effects of that complication were so uniformly unfavourable he proposed to say a few words about the two patients who recovered. An old soldier of plethoric habit with definite alcoholic history was admitted in January, 1901 with pneumonia of the right base. (The notes referring to that period had unfortunately been lost.) He was sent to the convalescent home in April, coughing up daily about 3 ounces of fœtid muco-pus. Whilst at the home he had several attacks of severe hæmoptysis. When re-admitted to hospital in July of the same year there was distinct clubbing of the fingers. He was anæmic from recent severe hæmoptysis. There were definite signs of a cavity of considerable size at the right base. The cavity was freely opened up and drained at the end of July by Mr. Pearce Gould. Early in October he was discharged convalescent, the wound having completely healed and all signs of gangrene disappeared. He remained in fair health until May of the following year, when the sputa again became slightly offensive and he had a moderate hæmoptysis. Five days after admission he had a severe rigor, followed next day by the expectoration of some very offensive muco-pus. A week later cerebral symptoms set in with frequent vomiting, death taking place at the end of three days. There was general purulent meningitis and a small abscess in the left occipital lobe. The right lung was very adherent and partially collapsed, and there was considerable dilatation of the extremities of some of the bronchi. No gangrene was present nor could any trace be discovered of the gangrenous cavity laid open the previous year. The interpretation of the clinical features of that case was admittedly complicated by the co-existence of bronchiectasis, which was the determining cause of the meningitis, but he thought it might fairly be claimed as a surgical success in the treatment of a gangrenous cavity of the lung. Bronchiectasis was not present previous to the original attack of pneumonia. The striking clinical feature of that case was undoubtedly the repeated severe hæmorrhages. The last case he would refer to was that of a heavily-built, pasty-faced, alcoholic individual of forty-one who was suddenly seized with general epileptiform convulsions and admitted to hospital in a state of semi-coma on September 8th. General convulsions recurred at intervals during the first twelve hours. As he became more fully conscious on the fourth day he complained of acute pain in the right side, and on the following day there were definite signs of pneumonia of the right base. On the ninth day there was gangrenous fœtor of the sputum. There were numerous crepitations and bubbling sounds over the right lower lobe but no cavity signs. He improved gradually till October 5th when the temperature rose suddenly to 103° F. and he coughed up a considerable quantity of fœtid muco-pus. After that fœtor rapidly subsided. On the 11th he had a similar attack with suffocative cough which all but proved fatal and for two or three days he brought up large quantities of fœtid muco-pus. After that improvement was continuous and before many days he ceased to expectorate. No cavity signs were discovered at any time, but the breathing remained permanently weak at the right base. Towards the end of November he underwent a severe operation for an unreduced dislocation of the left shoulder and left the hospital in perfect health at the end of December. In the June following, eight months after the attack of gangrene, he was still in perfect health and able to do his work as a gardener. That was a good instance of spontaneous cure of a pulmonary gangrene, and he might add without exaggeration that it would be difficult to conceive a case more unpromising at the outset. A generally stimulating line of treatment was adopted in both cases with inhala-

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tions every two or three hours of a mixture containing equal parts of pure carbolic acid, chloroform and eucalyptol.

Dr. HADLEY thanked the Registrars of the London Hospital for the great labour they had expended in collecting the statistics. There were 2362 cases in the last ten years, but none of those in the statistics were over ten years of age. Therefore the figures differed very much from what they would have been if the whole hospital population had been taken. The figures were collected from the working classes and differed very much from some which he had relating to the well-to-do. It was noted that the disease was three times commoner among men than among women, a fact which he did not see mentioned anywhere, but which he thought the collected figures from all the hospitals tended to show. That difference obtained in all the age groups. But it was not true in the returns from the Children's Hospital, and he thought the difference was largely due to the extra exposure of the working classes among the male sex. The mortality increased with age, and as patients under ten were excluded, one missed the great mortality among those of tender age. He had divided the age groups into 10 to 20; 20 to 30; 30 to 40; 40 and upwards. The smallest mortality was in the youngest age group, 10 to 20, amounting to only 5 per cent., whereas in the older cases it was nearly 50 per cent. That was largely due to the fact that the complications were much more numerous in older people. Another interesting point was, that although the cases were much less frequent in females than in males, the mortality was greater in females, and that was borne out in most of the collected cases. It certainly was so in those which came from the London Hospital. Complications were most important in the group which meant antecedent conditions. They were often lumped together with the remark that the cases were complicated because they had heart disease, forgetting that although it was a complication and increased the mortality it was not a complication of pneumonia. The cases from the London Hospital were separated into those which were complicated by antecedent conditions, and those complicated by actual troubles occurring as a result of the pneumonia itself. Many of the complicated cases were those of antecedent conditions, and in the latter cases the pneumonia was much more fatal. The effect of alcohol was very definite, for while the death-rate in all cases was 23 per cent., that of the alcoholic cases taken separately was 66 per cent. He had separated out also antecedent cardio-vascular changes, including aneurysm, from those which occurred in the course of pneumonia or after it. Antecedent trouble of that kind raised the mortality to 46 per cent. But the largest death-rate of any, outweighing anything which occurred as a result of pneumonia, was chronic nephritis, the death-rate of which was 82·6 per cent. Chronic bronchitis, emphysema, phthisis, or old fibroid lung, only raised the mortality from 23 per cent. to 31 per cent. Of the complications which occurred as the result of the pneumonia, empyema, delirium and pericarditis were the most important because the most numerous. Gangrene, abscess, endocarditis and pericarditis were the most fatal. Some of them were interesting but he would not refer to them in detail. His experience was that it was difficult to diagnose pericarditis occurring in the course of pneumonia; it was most frequently recognised in the post-mortem room. It should be carefully watched for, as there was little chance of a patient recovering from it unless it were frankly diagnosed and opened up. The few cases which he had seen recover had nearly all been opened up and drained, or at least tapped. The effusion was a very large one. In children it was important to notice that the enlargement of the pericardial effusion was directly backwards or almost so, and it would give rise to dulness at the left base, very much simulating pleural effusion, and not showing much in front. Still, if there were marked dulness at the back (which might or might not be a pulsating pleurisy) it would arouse suspicion, and then perhaps the observer would be able to appreciate the covered heart, the shifted apex and the muffled sounds indicative of pericarditis with effusion in front. But when the patients were very ill it would be difficult to diagnose the pericarditis. He believed many of the cases of supposed abscess of the lung which recovered were not really abscess at all. Once or twice he had seen an interlobar empyema which burst into the bronchus and was coughed up, and it was diagnosed as abscess of the lung. He believed

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real abscess of the lung, which must be localised gangrene of the lung, was very fatal. Sometimes a case had the following history: Pneumonia running the ordinary course, a crisis, but the temperature tending to become irregular again and then becoming of the hectic type. The patient did not progress, the signs remained, cough came on more markedly, and in a fit of coughing a good deal of pus would be brought up, after which the patient would get well. Because the amount of pus was not large the inference was that it was not empyema, but in many interlobar empyemata the quantity of pus was not great. From their position in the lung, between the lobes, they naturally impinged on the bronchus, often perforated that part and were coughed up. In the London Hospital there were eight cases of abscess, several of which got well, but if they had been really cases of abscess he did not think they would have got well. The symptoms of meningitis were so constantly seen as a result of toxæmia that one must be on guard when seeing the symptoms of meningitis in pneumonia. He had seen retraction of the head, headache, vomiting, irregularity of pupils, squint, fits, twitchings; he had also seen mastoids explored with negative results. With regard to peritonitis and colitis, abdominal pneumonia was a condition which must be recognised. Fellows were familiar with appendicitis admitted on the surgical side, which were, or were not, operated upon there, and then passed over to the medical side with pneumonia. There were also cases in which there had been acute peritonitis, in which the abdomen was opened and searched, but nothing found except fluid, and which, having been sewn up and passed over to the medical side, had shown definite signs of pneumonia a day or two after the operation. Pneumonia sometimes occurred in an epidemic form in institutions where the cases could be watched, and in such cases one type would show enteric symptoms and the other pulmonary symptoms. He had seen that in a village in the country, where the practitioner remarked that half the cases were typhoid and the other half pneumonia. In one house in that village there would be a case with acute tonsillar trouble and bronchial catarrh; in another case the symptoms of typhoid, and perhaps a third case would be one of typical pneumonia. In the statistics of the hospital he could not bring that out, because there were figures relating to various doctors during the last ten years, but which should suggest itself to the practitioner when he saw colitis or peritonitis and other complications of pneumonia. That led him to speak of abdominal distension, which had been referred to already. It was stated that in some cases it was due to paralytic, probably toxic, distension of the stomach. It had been seen in cases other than those of pneumonia, and he wished to emphasise the point that it was not always of the stomach, nor were the fatal cases always those involving the stomach, but that the intestines were also affected with toxic paralysis, causing great distension, which was a very bad sign, for the diaphragm was so much pushed up as to cause heart failure very quickly. He had passed long tubes from the point of view of treatment and had many times left a long tube in, and the patient passed flatus fairly readily with a diminution in the size of the abdomen. Most of the cases of arthritis in association with pneumonia were in young subjects, many of which were, naturally, not included in the present series. In regard to fibroid trouble, most of the cases were under the heading of antecedent conditions. It was not common to find fibroid conditions as a result of lobar pneumonia, most of the fibroid cases being the result of broncho-pneumonia as the result of measles or whooping-cough, or both, in early childhood. Where fibroid the result of pneumonia occurred, apical pneumonia was the commonest not to resolve, and in nearly all of them there was a strong alcoholic history. He could not say much about inoculation, because very little of it had been done at the London Hospital. But he gathered from others that there were many difficulties; the cultures did not live, and the acuteness of the disease made it difficult to get cases early enough in hospital. The crisis sometimes occurred on the second day, and it would be impossible to get a vaccine ready in that time from the patient himself. Even if that were done there would be a tendency to say the crisis was due to the injection and not to natural causes. The more debatable cases would be those in which there was some chronic manifestation as a result of the pneumonia, such as arthritis, empyema and non-resolution. In one case at Victoria Park there was an empyema of four weeks' standing, which would not heal. It was drained in the

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ordinary way, and Dr. Ross made a culture and vaccine, and injected it into the patient. The condition afterwards healed in about four days and the patient got well. He believed cases of arthritis would be very amenable to such treatment. Empyemata seemed doubtful, because in the majority of cases in which healing did not occur it was probably due to a double infection. In non-resolution although the resolution might be helped by the injection, it was very difficult to exclude the possibility of double infection, when no good effect from the injection of pneumococcic serum would be likely. He had always felt that pneumonia, rheumatism and tubercle might be placed in the group of diseases which predispose to a second attack rather than confer immunity. So that any immunity conferred in those conditions must be very short. In that class of cases it was necessary to move slowly.

Dr. DALTON said that in the statistics prepared by the Registrars at King's College Hospital for the purposes of this discussion, there were certain cases which ended by lysis, and he looked through the notes of those cases. There were 196 cases which recovered, and from these he excluded those in which there were obvious and gross complications, which would of course alter the temperature chart. There remained eleven cases in which the temperature fell by lysis, and it was those cases which he looked into. On investigation he found that in nine cases the signs and symptoms were ordinary and typical, but the temperature all through was remittent, so they were not typical cases in respect to the course of the fever, and it was not surprising that they should terminate by lysis. He had no doubt that such remittent temperatures were due to some latent complication, or some secondary infection, which interfered with the ordinary course of the pneumonia. There were two cases left over, and in these even the temperature chart was typical, except that defervescence was by lysis. One of these cases he could not explain, but the other was under his own care, and showed that in it there was not true lysis. It was in every way a typical case, with consolidation at the right base. On the fifth evening the temperature began to fall, and took thirty-six hours to reach the normal. It then rose again to 100·6°F., and the rise was accompanied by some collapse, with fresh cough and bringing up of blood. Then some physical signs were found in the left lung. Therefore it seemed to be a case of true relapse rather than of lysis, a fresh attack of pneumonia affecting the other lung at the time when the crisis of the first attack was in progress. He thought dilatation of the stomach very common in pneumonia, and the point had been raised by Dr. Pasteur. But the distressing condition of the patients in so many cases prevented that point from being investigated. He thought in influenza very great dilatation of the stomach was apt to be present in those cases where the influenza affected the alimentary canal.

Dr. GOSSAGE said he thought it a pity that the statistics were limited to patients over ten years of age. Out of 577 cases at Westminster Hospital, 246 were under the age of ten years, and 246 was really below the number, because in one or two years all the cases under three or four years of age had been included as broncho-pneumonia, regardless of the clinical aspect of the case. Therefore lobar pneumonia was as common in children under ten as in persons over that age, or commoner. The diagnosis of lobar pneumonia in children was less certain than in adults, because of the difficulty of distinguishing between broncho-pneumonia and lobar pneumonia, and physicians differed very much in what they regarded as the clinical manifestations of lobar pneumonia. Frequently, too, in the post-mortem room the diagnosis which had been made was found to have been wrong. Quite apart from that, however, it was obvious that lobar pneumonia was very frequent in childhood. The complications affecting other parts were more important in children than in adults. He took empyema as an illustration, that being the commonest complication. Empyemata were open to the objection that they were collected not only from the cases which arose in the hospital, but from those in which the disease commenced outside, but in a comparison between different ages this did not matter. The proportion of empyemata to total number of cases of pneumonia at each age was nearly equal for all ages. There were a large number of empyemata which proved to be pneumococcic, but in which there was no history obtainable of previous pneumonia, and no doubt many of these cases of

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empyema had had pneumonia beforehand, although there was no history. These cases were more numerous in children, and therefore the proportion of cases of empyema in children was rather higher than would be supposed from a study of statistics. The percentage of deaths from complications in children, compared to the total deaths from pneumonia at each age, was much higher than in adults, in fact death from pneumonia between two and ten years of age, provided the child was healthy beforehand, was almost invariably due to one of the complications. He had worked it out in regard to the cases under ten years of age, at Westminster and Shadwell, and out of 1005 cases of lobar pneumonia there were ninety-five deaths, and of those ninety-five, sixteen when they were attacked with lobar pneumonia were suffering from some other complaint, and death resulted because their resistance was lessened. Eliminating these, there remained seventy-nine cases of what might be termed healthy children when they were attacked. Of those seventy-nine, fifty-one deaths were due to some pneumococcic complication. There were forty-one cases of empyema, eight cases of meningitis apart from empyema, one case of purulent pericarditis apart from empyema, and one case of pneumococcic clear serous effusion. That meant that 65 per cent. of the deaths occurring between the ages of one and ten years were due to some other pneumococcic infection. Under the age of two years, toxæmia was a great factor in causing death, and in the statistics furnished by Shadwell there was a considerable death-rate under one year of age apart from complications. Another factor, which was almost the most important in producing a larger death-rate at earlier ages, was, that lobar pneumonia tended to be complicated by broncho-pneumonia. It was difficult to define exactly what was the distinction between those two conditions, and a large number of cases of broncho-pneumonia were due to the same specific organism as lobar pneumonia. One also found that the complications of broncho-pneumonia were the same as those of the lobar form of the disease: there was the same tendency to the pneumococcic infection of the pleura, the peritoneum and the meninges, though the proportion was very much less. It was not surprising, therefore, that secondary infection with broncho-pneumonia was the cause of a considerable amount of the higher death-rate in very young children.

Another important point in regard to the statistics was the very large number of cases of apparently primary pneumococcic empyema. Probably a selection of those had had pneumonia beforehand, but there seemed to be a large number of them, and the history was fairly clear, allowing for the non-intelligence of parents in some of them, that there did not seem to have been any sign of lobar pneumonia before the development of the empyema. If only some of these cases were primary, that would make the pleura a seat of selection for primary infection by the pneumococcus, secondary only to the lungs and the middle ears. It was known that the pneumococcus could affect the pleura primarily, just as it did the pericardium, and peritoneum, and meninges, for there was a particular type of infection of the pleura, a very acute type of empyema, which generally produced death very rapidly. But these cases under consideration were usually ones which had had empyema for a long time, and which came into the hospital for operation. The deaths from such were no more numerous than in cases which occurred after pneumonia.

Another point was that the pneumococcus did not necessarily bring about a purulent condition. One found that a certain number of cases of clear serous effusion into the pleura were due to the pneumococcus, and yet never became purulent. There had been one or two cases of that kind at Shadwell. One case, however, subsequently did become purulent. That was of some importance with regard to the possibility of there being a pneumococcic meningitis which did not kill. Dr. Hadley had pointed out how frequently there might be symptoms of meningitis, and the majority of cases of this kind cleared up all right, and probably never had had meningitis at all. Some might merely have had otitis media. In one case, however, at Shadwell, the child recovered, though it had squint, head retraction, twitchings, and optic neuritis, symptoms very suggestive of meningitis. So it was not improbable that pneumococcic infection of the meninges which did not go on to the formation of pus might occur very rarely and clear up without causing the death of the patient.

Dr. ARTHUR LATHAM said the statistics of St. George's Hospital showed the importance of the part played by exposure in the induction of pneu-

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monia. Of 634 cases of persons over ten years of age, 526 were male and 108 female; in other words 83 per cent. were male and only 17 per cent. female. He thought it probable that in males exposure was a much more constant factor than in females. That was also borne out by the fact that in the curve of incidence in females the greatest incidence was at the age of thirty-five, *i. e.* at the age when exposure might be assumed to play its most important part. The curve of monthly incidence reached its maximum at the end of May and its sub-maximum at the begining of November or the end of October, which was the time of year when, in the one case clothing was too early left off, and in the other winter clothing was too long delayed. Dr. Robinson, who had recently returned from Bulawayo, had told him that the hospital there was practically empty except at two periods of the year. In the hot rainy season it was filled with cases of malaria, and in the cold season with cases of pneumonia. It had been said that in the majority of cases of pneumonia the knee-jerk was absent. They had paid attention to this point at St. George's in 125 cases, and found that in 90 of those the knee-jerk was brisk, and in the other 30 it was either sluggish or absent. Another fact which had been brought out by Dr. Golla, of St. George's, was that the nasal respirations and the abdominal and thoracic respirations did not show precisely the same curve; at times the curve of nasal respirations was distinct from and independent of the thoracic curve. The same was true of the abdominal respiration curve. It was stated in the returns from St. George's Hospital that pneumococcic serum had been used in four cases, with a mortality of *nil*. One of those cases was that of a man, æt. 21 years, who was admitted with consolidation of the lower left lobe, and on the seventh day the temperature fell from 103° F. to subnormal. It was thought that this fall was due to the crisis. The following evening the temperature again rose to 102·8° F., and then there were definite signs of consolidation at the base of the other lung. There had been no evidence before of the other lung being involved. Twenty cubic centimetres of anti-pneumococcic serum were given, which was obtained at the Lister Institute, and in twenty-four hours the temperature had become normal; it remained normal and the man made a rapid convalescence. In some cases anti-diphtheritic serum had also been tried, and the reason for that was because of the apparent effect that it had produced in a case of broncho-pneumonia. A girl was admitted under his care during the present year at the age of three and a half. She had been ill for twenty-four hours before admission. The temperature was 102° F., and there were signs of broncho-pneumonia. There was a history in the family of diphtheria. There was extreme difficulty of respiration, with recession of the intercostal spaces, and he thought that there was obstruction of the larynx due to the presence of a membrane. But there was no sign of diphtheria, and no membrane to be seen. It was thought best to give the child the benefit of anti-diphtheritic serum, and 4000 units were injected at once and 6000 units a few hours later. In thirty-six hours the temperature had become normal, and the child, who at first he thought was likely to die, made an uninterrupted recovery. Careful examinations were made of the material obtained from the fauces and from the nose, but no diphtheria bacilli were discovered. Another child, æt. 16 months, was under Dr. Rolleston, with a temperature of 104° F., and an illness of fourteen days' duration, suffering from broncho-pneumonia. The patient received 5000 units of anti-diphtheritic serum; the temperature fell next day and in twenty-four hours it became normal, and remained so till the child left the hospital. There was no evidence of diphtheria. On the basis of those cases, both of which were severe, and in both of which, coincidently with the introduction of anti-diphtheritic serum the temperature fell, it was thought advisable to try the effect of that serum in certain cases of pneumonia. A girl, æt. 6 years, was admitted with a temperature of 102° F., with consolidation in the right upper lobe, and with some bronchitis in the left lung; 6000 units of anti-diphtheritic serum were given, and the temperature fell the following morning and remained normal. A woman, æt. 36 years, was admitted on the third day of the disease with consolidation of the left lower lobe, and a temperature of 101·8° F.; 12,000 units of anti-diphtheritic serum were given and the temperature began to fall, and on the morning of the second day it was normal and remained so. Another patient was a woman, æt. 38 years, who was admitted on the fourth day of the disease, with a temperature of 104° F. There was consolidation of the lower left lobe and one of the left upper lobes; there was also a systolic mitral murmur: 12,000 units of anti-diphtheritic serum were given and the temperature fell slightly, but did not become normal until the eighth day of the disease. In one case 20 c.c. of fresh horse serum were given, but in that case there was no particular result. He had been asked to bring forward those records, though he did not suggest there was anything more than a coincidence in the effects observed after the use of the anti-diphtheritic serum. But he called attention to the fact that all the cases dealt with were severe ones. The serum had been given only in those cases in which the prognosis seemed to be grave, and in no case in which the serum was given had the temperature reached the point at which it stood before the administration of the serum.

(*To be continued.*)

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A CLINICAL LECTURE

ON

A CASE OF MUSCULAR ATROPHY.

Delivered at St. Bartholomew's Hospital.

By W. P. HERRINGHAM, M.D., F.R.C.P.,
Physician to the Hospital.

GENTLEMEN,—The case I show you is that of a Hebrew boy, æt. 17 years, who was admitted into this hospital in August. His story was that he had been ill a fortnight, and had suffered during that time first with tonsillitis, ordinary sore throat, and secondly with fleeting pains in many different joints. When he came to us he had slight fever and swelling of one of his wrists. There was no doubt about the diagnosis of rheumatic fever, and there is none now. But he had never had rheumatic fever before, and as he was at this time seventeen years of age he remained, as is not uncommon, free from heart disease. We put him for two days upon salicylate of soda, in rather large doses, and by the end of that time he was free from pain and fever. We supposed he was in the way of convalescence, but in a few days he had a return of pain in his right shoulder, such as is often experienced when the patient is not fully cured. He did not move his right shoulder, which, considering the pain, was natural enough. That joint is so deeply seated and so thickly covered with muscles that it is practically impossible to examine it very thoroughly. You cannot tell whether there is slight effusion there, and the redness which you can often see in the more superficial joints does not come through to the skin in the case of the shoulder-joint, because the tissues are so thick, and so there is nothing but pain to tell you what is going on. I supposed it therefore to be an ordinary relapse of the rheumatism. But in two or three days' time it was pointed out to me by the

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clerk of the case that he had considerable wasting of the deltoid muscle, and within a little time of that he was well enough to be tested in regard to his electrical reactions. And then, even at this early day, it was found that he had almost complete reaction of degeneration in the deltoid muscle, and some weakness in the other muscles about the shoulder-joint, for instance in the brachialis anticus and the biceps. In a few days, instead of getting better all these symptoms increased very much indeed, and within a fortnight of the time that he was taken ill with the pain in his shoulder he had almost complete loss of power in all the muscles about the shoulder-joint, and great wasting of those muscles and the muscles of the arm, with considerable weakness and some wasting of the muscles of the forearm, both front and back. He also had complete reaction of degeneration in the deltoid, biceps, brachialis anticus, in the supra- and infra-spinati, and some loss in the muscles below the elbow. There was, besides, great pain and tenderness with some thickening of the musculo-spiral nerve, where it comes round the border of the humerus, and when I passed my fingers up the axilla and compressed the brachial plexus against the bone he felt considerable pain. And besides the pain and thickening of the nerves he had also anæsthesia, the chief part of which was on the outer side of the arm and the pectoral border of the axilla, but which in a less degree occupied almost all the limb. With the exception of the loss of pain and a considerable recovery from the anæsthesia he has remained almost in the same state. He is now getting a little muscular power back, there is more power in his hand, and he can grasp better than he could. And he can move a little the muscles which bring about pronation and supination. There is practically no power of flexion of the elbow, nor, though passive movement is free, is there any voluntary movement of the shoulder, but he has slight power of extension of the elbow. There is much wasting of the muscles about the shoulder-joint, as is very plainly visible to you. I show you a diagram which was made of the extent of the anæsthesia, which will show you what it was like in the worst stage.

Now came the question as to what was the matter with the boy. The principal symptom in the case was the extreme paralysis and muscular wasting. Muscular wasting arises, in the first place, from diseases of the muscles. There are certain diseases of muscles which, from some developmental fault in the muscular tissue, lead to wasting, and, consequently, paralysis. Those are generally called dystrophies or myopathies, a name which implies that it is rather a fault of development than a secondary atrophy of muscle, the word atrophy being applied to muscular wasting which comes on as a result of some other lesion. Those dystrophies are characterised, you may say, by three points. In the first place they are very chronic, in the second place they are very apt to be hereditary, and in the third place they follow certain definite groupings, which are fairly well worked out, and of which the one you see most commonly is pseudo-hypertrophic muscular paralysis. But they are quite separate and distinct from a case like this, in the following manner: that they are never accompanied by any pain at all nor does the reaction of degeneration come on, as it did in this boy, rapidly. Such alteration as occurs in the electrical reactions occurs gradually, and is then proportionate to the gradual wasting of the muscles. So it could not be that. Considering the nervous symptoms in the case, we felt obliged to conclude that the muscular atrophy was due to some nervous disease.

In the first place, in considering where a nervous disease causing muscular atrophy is situated you can rule out the brain. It is a broad and safe rule that diseases of the brain do not cause muscular atrophy. There are a few cases in which in hemiplegia you get a certain amount of atrophy and a certain amount of electrical alteration. But it is very slight; it is not in the least comparable to a case like this. You may practically, therefore, say as a general rule that muscular atrophy never means disease of the brain. It may mean disease of the spinal cord, and if so it is disease of the great cells in the anterior cornua and the tissue surrounding them. That is one common cause of muscular atrophy, and disease in that region occurs in two forms. In the first place it occurs in what we call infantile spinal paralysis. Although it is called infantile you must remember it does occur occasionally in adults, especially young adults. Though hardly any cases occur after eight or ten years of age, you cannot say from the fact of the patient being beyond childhood that it cannot be infantile paralysis. In some ways the onset of

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the disease was not unlike that of infantile paralysis. In a case of infantile spinal paralysis a child is ill with pain and fever for a couple of days or so, and it is after that you notice that the limb or limbs—for it often attacks three or four limbs—are weak, and even, perhaps, have complete loss of power. It may be termed a massive and sudden paralysis, preceded by a couple of days' fever and pain. That was not unlike this boy's case. But in infantile spinal paralysis you never get tenderness down the nerves, with thickening of them, and you never get a persistenta næsthesia. There is that rapid wasting and the reaction of degeneration, but sensory symptoms, except the partial pain, are absent. So we could say at once that it was not a rare case of infantile spinal paralysis occurring in a young man. Anterior cornual disease occurs also in the chronic form, and forms a group of cases in which lie progressive muscular atrophy, amyotrophic lateral sclerosis, and syringomyelia. But in all those cases in the first place the disease is very chronic and begins very gradually; secondly, it does not begin near the trunk, but in the periphery, such as in the hands; again, it is nearly always symmetrical, though I have recently had a case which is an example to the contrary, in which the whole of the left arm was affected, but the right not at all; and lastly, in no member of the group is cornual disease accompanied by such pain as this man had, nor with tenderness and swelling of the nerves.

That leaves us, therefore, with the spinal nerves as the only possible nervous cause of such a case as this. When the nerves involved belong to a plexus, the brachial or the lumbar, you ask yourself whether the symptoms point to an affection of the roots which form the plexus or to an affection of the nerves below the point of junction? And when I showed this case in consultation in the theatre the first alternative was suggested. For it so happens that there is a disease very like this which is known to be occasioned by injuries of the spinal roots in the neck at the time of birth. It is called Duchenne's or Erb's paralysis, or Duchenne's birth palsy. I show you the picture of such a case, because it occupies exactly the same situation as that which is principally affected in this boy's case—that is to say, the supra- and infra-spinati, the deltoid, the brachialis anticus, and biceps, and the supinator longus. The suggestion made me look very carefully at the spinal roots. But in the first place if it were a disease of spinal roots every one of the spinal roots going to the plexus must have been affected, for the anæsthesia and motor symptoms were spread over every one of them. Secondly, on examining the neck as carefully as we could there was no tenderness in the spinal roots, nothing abnormal could be felt in the neck, nor was any disease of the cervical vertebræ shown by the X rays, so I felt certain it was not a disease of spinal roots, and that left the nerves of the brachial plexus as the only source of origin. Neuritis seemed to be the only possible diagnosis to explain the case.

What do you mean by neuritis? Neuritis is of two kinds: There is what is called parenchymatous neuritis; there is another kind which is called interstitial neuritis. Parenchymatous neuritis is a condition of which you see examples fairly commonly. Lead paralysis is almost certainly a parenchymatous neuritis. Alcoholic paralysis is another; arsenical paralysis is another; diphtheritic paralysis is another. And you find similar paralyses occurring after other fevers, sometimes after influenza, sometimes, but very occasionally, after typhoid. And if you think of all those paralyses you will see that in the first place they seem all to be due to some form of poison, that is to say, they are all toxic in their nature so far as their pathology goes. Secondly, so far as their symptoms are concerned they have all got a rather typical seat of predilection. The parts which each kind of paralysis affects are generally the same. For instance, in lead palsy there is the wrist drop, in alcoholic paralysis the lower limbs are affected and there is the foot drop. In diphtheritic paralysis the legs and afterwards other muscles are affected. Arsenical paralysis affects the same places as alcoholic. They all seem to have a certain definite area which they affect first. Thirdly, they are nearly always symmetrical, affecting both limbs, so that that class of disease is sometimes called symmetrical neuritis. And in the fourth place, nearly all the cases show a much greater involvement of motion than of sensation. There is no doubt that there is a certain amount of pain experienced in these cases, but that is usually much less in degree than the amount of weakness which is present.*

There is another class of cases, which is the opposite of these, namely, interstitial neuritis. In this form the disease is nearly always local, and it is nearly always exceedingly painful, and affects sensation much more than it affects motion. Examples of this are common enough. Sciatica is the principal and best known. There is just a similar sort of affection in the nerves of the

* I did not mention Tooth's paralysis for fear of confusing the students; its pathology is very difficult.

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arm. Very often the nerves which go down the extensor surface of the forearm, apparently that posterior branch of the musculo-spiral which goes round the radius, gets affected. I have had it myself, and I have known many people who have had it.

That part of the arm gets so tender that the arm is practically useless, the pain being caused by the contraction of the muscles on the nerves. You would scarcely think that contraction of muscles could cause so much pain. And that leads on by a natural gradation to other things, such as writer's cramp, and piano-player's cramp, in which it is not exactly over-use of the muscles, but only certain uses of the muscles which produce pain and stop movement. For instance, it is only when the person plays the piano that he has to stop, and if writer's cramp is the trouble most other movements besides writing can be performed. I have known the same thing occur in other trades. A meat carver came to the hospital when I was assistant physician, and said he could do almost anything except carve, but that that action hurt him so much that he had to stop. I do not suppose any of these patients have the full use of their muscles, but there is nothing which they are actually stopped doing except the particular action of which they complain. I think that is simply because some nerve is pressed upon by the particular group of contractions special to the occupation, and that no other occupation hurts the nerve, at any rate to the same extent, because it does not involve the same group of contractions. But I believe these "cramps" are the same sort of thing as the ordinary brachial neuritis, and that in them, as in the latter, the pain is caused by pressure of contracting muscles.

There is another group of cases that I think is of precisely the same nature although they are given a different name, and are called muscular rheumatism. Examples of that are lumbago, "deltoid rheumatism," which we so often see, stiff neck, and "rheumatism" about the back of the scapula and the muscles at the back of the neck. All those, I believe, are of the same description, an interstitial neuritis of small nerves.

Interstitial neuritis is characterised by thickening and tenderness of the nerves themselves with great pain, the pain being referred to the various points of distribution of the nerve. Examination of the affected nerves easily explains the symptoms. You know what a nerve is like; it consists of a thick fibrous sheath in which are bundles of nerve-fibres, each bundle having its own fibrous sheath enclosed in the big one. Each bundle is also permeated by connective tissue running between the separate nerve-fibres which form it. When you examine the sciatic nerve in a case of sciatica, as has been done occasionally, you find that it is thick, just as we found this boy's musculo spiral nerve when we felt it the other day. It is also pink from inflammation instead of being a glistening white. And if you cut into it a lot of exudation escapes—exudation of gelatinous character which is enclosed in the tight fibrous envelope. That is quite sufficient to produce the pain, partly by pressing on the little sensory fibres which go to the sheath of the nerve itself, and partly by pressing on the sensory fibres of the main nerve. And there is no doubt that pain is caused by both of these, because the pain is referred not only to the place itself but also to the point of distribution of the sensory branches of the nerve. The proof that the same thing happens in the cases of muscular rheumatism is in the clinical connection between the two. I know, from painful experience, that lumbago passes continuously into sciatica. And you will find that that is repeatedly the case in a good many patients. I know, too, very well, that the painful points in the muscles about the scapula and shoulder closely correspond to the points of exit of the sensory nerves where they come through the fascia, and that is exactly what occurs in sciatica, where the painful points in the leg are the points at which the superficial branches come through the deep fascia. That is very well known, and those things make me think that the disease is the same in the two cases; in the one case there is an affection of a great nerve trunk and in the other of the small nerves supplying muscles or skin.

How does such an inflammation begin? Sometimes the sheath of the nerve is first affected. For instance, in a traumatic case that would be true, and perhaps in writer's cramp and such conditions, where the pain is caused originally by contraction of muscles on the nerve. It is true in a bullet-wound case, or in injury to the neck at birth; these are all cases of disease beginning in the nerve sheath. But in the great majority of the cases I think the inflammation spreads from fibrous tissue in the neighbourhood. I believe that to be the case in lumbago and sciatica; that it is the same sort of inflammation which, for want of a better name, we call a rheumatic affection of the fibrous tissue, first squeezing the little end nerves in that tissue, and afterwards spreading to the main sheath of the nerve. That is also the case in another form of local and interstitial neuritis, which is not of a sensory nerve, and that is facial palsy such as we so commonly see here. Bell's paralysis, as it is called, is frequently caused by disease of the middle ear, affecting the temporal bone and inflaming or pressing upon the sheath of the nerve in the aqueduct of Fallopius. That is especially interesting for this reason: that although it is purely a motor nerve, yet there is slight pain and tenderness in it, though not such intense pain as is usual in sciatica. That is because the little sensory nerves which go to the sheath of every nerve are affected by the exudation which takes

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place around or within the sheath. The nerve itself is insensitive because it is a purely motor nerve. There is another very interesting example of that sort of neuritis in what I hope will be called Dr. Lewis Jones's neuritis, because he has illustrated it so well, the neuritis that is due to an extra-cervical rib pressing on the lowest roots of the brachial plexus. It causes paralysis, atrophy, and great pain. And the same sort of thing, I believe, happened in this particular boy: the neuritis from which he suffered was due to a spreading of the inflammation from his rheumatic shoulder. We know what a rheumatic joint looks like when it is superficial, as, for instance, the ankle, or wrist, or finger-joint. It is swollen, and that is due partly to synovial effusion, but also partly to thickening of the capsular and other ligaments round it. And the inflammation goes right through those structures and comes out at the skin, producing redness and swelling of the skin itself. So it is an inflammation which spreads a good way beyond the synovial membrane of the joint and affects all the structures in the neighbourhood. Now, the brachial plexus of nerves lies very near the shoulder-joint. The tendon of the subscapularis, which lies between, is incorporated with the capsule of the joint, so that the inner surface is lined by the synovial membrane of the joint. Besides that, this bursa, if enlarged, comes down below the lower edge of the capsule and can be exposed without taking away the subscapularis at all; so the nerves are lying closely in relation with the inflamed joint. More than that, the very nerve which was first affected, the nerve to the deltoid, is the circumflex nerve, which runs through the quadrilateral space and actually touches the capsule of the joint in that space. That was the first nerve affected and the worst affected for a considerable time. Other nerves in the neighbourhood had the affection later. So I think it probable that the inflammation of the joint spread to the nerves lying in neighbourhood. And there is another thing to be said. You notice that the supra- and infra-spinati are affected, but that cannot be explained by the affection of any nerve close to the shoulder, because the nerve to these muscles is the supra-scapular nerve, which is given off in the neck. But just as inflammation clearly spread down the musculo-spiral nerve, so that we found it thickened and tender above the elbow, so we must allow that it may spread up the same nerves into the neck, where the supra-scapular is given off. And that, I think, explains how a nerve given off above the shoulder-joint was also affected.

In a case of that sort what treatment is likely to do any good, and what is likely to be the result? The first thing when a nerve is acutely inflamed is to keep it at rest. Do not try anything at all at first, except anodynes. Heat is one of the best. And opium lotion is very good, so are anodyne liniments, of which one which I have found excellent is a solution of menthol in a soap liniment, thirty grains of menthol in one ounce of linamentum saponis. Counter-irritants, blisters or the cautery, are sometimes very useful. But they cannot well be applied in the axilla. Beyond that you can do nothing while the nerve is acutely inflamed. After that is over and you are left merely with the results of the exudation, then you can hasten absorption, first of all by applying electrical currents. This boy is having electrical treatment at present. I do not think there is any drug which hastens absorption, but I have known aspirin relieve pain when nothing else would. You can also do good by the local application of heat, especially in the form of baths, for instance, at some of those places where they have well regulated douches, as at Bath and Aix-les-Bains. Such treatment, however, does not always do them good. When I was still very lame after sciatica I went to Algeria, and there I came across boiling springs which had been turned into baths by the Moors. I had a jet of water as hot as I could bear it on my sciatic nerve for twenty minutes at a time, so that in a week I had a pigmented patch in that situation. But I do not think it benefited my lameness at all. Still, some cases are cured by that means. Lately a lady who was a patient of mine had an attack of brachial neuritis when she was in Italy, and she went to baths near Padua, mud baths, where there are also hot springs. There she was partly treated with the ordinary vapour baths, and also her arm and side were packed in the hot mud. In a very little time she was completely cured of her pain, and able to use her arm. So I fancy that must be a better way of applying heat than ordinary baths. These remedies are chiefly for the relief of pain.

So far as the muscular atrophy goes, in most cases of local neuritis the prognosis is very good. In sciatica there will often be considerable atrophy, to the extent of one or two inches in the thigh and half an inch in the leg, but it will get completely well again as the exudation is absorbed and the nerve freed from pressure. I think the prognosis is much more doubtful when there is such extreme atrophy as is present in this boy. He still has the reaction of degeneration in all the muscles about the shoulder. I confess I think the result is very doubtful. But it never does to despair, and Dr. Lewis Jones has had so many remarkable recoveries from muscular atrophy after many months of electrical treatment that one should never say to the patient that the disease is irrecoverable. One should encourage the patient, and say, you never can tell how long atrophy of muscles due to neuritis may go on, and yet in the end be cured by perseverance in that treatment.

January 13th, 1908.

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THREE LECTURES
ON
THE SURGICAL DISEASES OF CHILDREN.* †

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LECTURE II.

GENTLEMEN,—I propose to-day to continue the remarks on the diseases incidental to birth. Although what is said may not be regarded as entirely surgical in its bearings, still the matters are of considerable importance, and therefore I shall go into them fairly fully.

The first affection is *icterus neonatorum*, or the jaundice of newly-born children. This is not at all an uncommon condition. I have no doubt that something like 60 to 80 per cent. of children after birth present signs of jaundice. And in a very large proportion the jaundice passes away without leaving any traceable ill-effects. But in some cases the jaundice deepens, and then it becomes a sign of very serious import. It is a striking point with regard to the jaundice of children who are newly-born, that unless it is of a very intense description the conjunctivæ and urine are not coloured; whereas in icteric adults there is always a yellowness of the conjunctivæ and an alteration in the colour of the urine. There is no doubt that jaundice is often present in newly-born children, but it is not suspected, and escapes observation because it is difficult to detect, unless you employ the test of pressing your finger on the skin, when the impression which is left is seen to have a yellowish tinge. As I have said, in a large proportion of the cases which are affected at birth, the jaundice passes away and leaves no sign. No doubt the slighter cases are merely due to the breaking down of a certain amount of blood pigment, which gives rise to the yellowish appearance. But the more serious cases are generally associated with septicæmic changes, or with syphilitic infection, or due to abnormalities of the bile-ducts. These are the main causes of serious and often fatal forms of jaundice. You also find jaundice in the newly-born, associated with acute fatty degeneration, and sometimes with what is known as Winckel's disease, which I shall mention presently. It is also seen to some extent in that curious condition, sclerema neonatorum. With regard to septicæmic jaundice, it is due to the general breaking up of the blood-corpuscles which takes place in septicæmia.

Next, with regard to the influence of hereditary syphilis, the underlying cause of the jaundice in that case is undoubtedly syphilitic peri-hepatitis, that is to say, a thickening of the fibrous tissue around the liver, with extension of the process into the liver substance. While I am speaking of that subject I want to give you a word of warning as to the diagnosis of hereditary syphilis. A child may be born apparently healthy, looking well in every respect, without any rash or any evidence of syphilitic infection. But in the case of a good number of children who die shortly after birth you will be able to find, if you look for it, distinct evidence of congenital syphilis, among the evidences being peri-hepatitis. There are also interstitial changes in the kidneys and in the spleen, and pneumonia alba is present; and these are quite sufficient evidence of this affection. The congenital abnormalities of the bile-ducts are interesting, and they may be limited to the common duct, to the cystic or the hepatic duct. In some cases the common duct is the subject of atresia just where it enters the intestine, and in other cases the whole of it is obliterated and there remains nothing but a fibrous cord. In yet other cases the cystic duct is either narrowed or entirely obliterated, or the hepatic duct is so affected. If the common duct is obliterated the gall-bladder is sometimes full of bile. It is noteworthy that in those cases in which the common duct has been obliterated the meconium itself is quite uncoloured.

Now, as you know, adult patients who are the subjects of jaundice are liable to hæmorrhages, and if by any chance a vessel is wounded it often happens that there is more hæmorrhage than normal, which may cause some anxiety, and you are likely to encounter after an operation secondary hæmorrhages. Therefore in an infant who is the subject of jaundice you may find multiple hæmor-

* Delivered at the Polyclinic.

† For information on many points in these lectures, the lecturer is indebted to Messrs. Ashby and Wright's excellent text-book on 'Diseases of Children.'

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rhages all over the body in severe cases, and punctiform hæmorrhages on the mucous membranes and beneath them, and even larger hæmorrhages in serous cavities. The children who are the subjects of these severe hæmorrhages generally die. Very few, indeed, of them recover.

The next subject which I shall take is *hæmorrhage* occurring in the newly-born infant. The characteristics of this hæmorrhage are, in the first place, that it does not appear to be connected with delivery; secondly, it is spontaneous; thirdly, it is multiple in situation; fourthly, it tends after a time to cease, that is to say, if the child live long enough; and lastly, it is almost always associated with some general infection. You may describe hæmorrhage occurring in the newly-born under two headings: (1) traumatic, (2) idiopathic. The traumatic hæmorrhage you will generally find is connected with the umbilical cord, whereas the idiopathic variety is associated with some general constitutional condition. The sub-divisions under which I wish to treat of hæmorrhage occurring in the newly-born are, hæmorrhage from the gastro-intestinal tract, hæmorrhage from the umbilicus, and hæmorrhages from the genitals. Hæmorrhage from the gastro-intestinal tract is due to many causes of which I must mention a few. You must exclude such sources of fallacy, especially if hæmatemesis be present, as the case of the child who sucks a cracked nipple and in that act swallows blood with its milk and afterwards vomits it. This may be very misleading, and induce you to suspect that the child is the subject of ulceration, of which I shall speak more fully presently. Again, children sometimes pass blood *per rectum* who have been the subjects of asphyxia neonatorum, which you will remember was dealt with last time. I alluded to it then as a factor of the gravest importance in the life of the infant, and I said the results of asphyxia neonatorum were very serious, and among them is this tendency to hæmorrhage. Such children may pass blood by the rectum in considerable quantity. But the most important cause, although it is the rarest cause of hæmorrhage occurring from the gastro-intestinal tract, is the presence of an ulcer. There are, to my knowledge, at least three cases recorded in which an infant died a very short time after birth from persistent melæna, and the cause was discovered to be an ulcer of the duodenum in each case. It was nothing but a simple ulcer, but it sufficed to kill the infant. And one may presume that if a child the subject of gastro-intestinal hæmorrhage, as shown by melæna and hæmatemesis, recovers, it has not been affected by a gastric or duodenal ulcer, because those conditions are so extremely fatal. You also meet hæmorrhage in other conditions which I may mention, such, for instance, as Winckel's disease, in acute fatty degeneration, and in acute œdema.

I will now pass to a consideration of the hæmorrhages from the genital tract. They generally occur in female children, and Dr. Cullingworth has collected thirty-two instances of hæmorrhage from the vagina occurring in babies. Unlike the last group of affections, it is generally recovered from. The explanation of the bleeding is not quite certain, but it seems to be dependent upon the presence of pelvic congestion which has resulted from the sudden obliteration of the umbilical arteries. It seldom occurs in male children, but it is comparatively common in female infants.

We must speak of the subject of umbilical hæmorrhage, which is an important matter. And I propose to divide it into what I may call traumatic and, for want of a better word, idiopathic. I make this distinction because the two conditions are totally different in every respect. But they are particularly so in the matter of prognosis. Subjects of traumatic hæmorrhage recover, whereas those children who have the idiopathic variety almost always die. Traumatic hæmorrhages may be traceable to the following causes: either the umbilical vessels are insufficiently ligatured, and the ligature slips off; or else what happens is that the vessels are tied too tightly with a ligature which is too thin, which cuts into the vessels, in consequence of which hæmorrhage sets in. Or, again, hæmorrhage from the umbilicus may be due to cutting the cord too short. We know that the drying of the cord proceeds from its extremity upwards, and it is also a gangrenous process, and if the cord is cut too short the gangrene proceeds too rapidly, and vessels are invaded before they have been properly sealed. Hæmorrhage from the umbilicus also may be due to the roughness of the nurse in knocking off the scab. But the matter is readily

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dealt with on ordinary surgical principles, and you must either ligature the vessel or under-run it with a threaded needle.

The other type of hæmorrhage from the umbilicus, the idiopathic hæmorrhage, is a very different matter altogether. It is seen to occur in foundling institutions. It often assumes a more or less endemic character. Another point is, that it is an oozing and not a steady flow from a vessel, and it is exceedingly difficult to stop. Then, again, it is connected with, and dependent upon, and often a symptom of a very serious general disease, such, for instance, as septicæmia; or it is associated with the presence of jaundice, or with other profound changes which I am about to mention. The hæmorrhage comes on about the fourth or fifth day and persists for some days, until either it stops and the child recovers, or what is much more common, namely, in about 80 per cent. of the cases, the child dies simply of exhaustion. The exact cause of it is, as I have already said, extremely difficult to ascertain. There have been certain researches made upon it, especially on the bacteriology. The organisms which have been found have been streptococci and staphylococci and pyocyaneus. Gaertner, some ten years ago, discovered in the blood of these cases a short bacillus which closely resembled the *Bacillus coli* (Ashby and Wright). Hall, in America, also discovered in the umbilical arteries in these cases a similar bacillus, and when this bacillus was isolated and cultivated and injected into guinea-pigs it produced persistent hæmorrhages, which were very difficult to arrest. Therefore it seems that the cause of this peculiar and persistent bleeding is probably what is known as Gaertner's bacillus, which may be a modification of the *Bacillus coli*, but often mixed with it are found the *Bacillus pyocyaneus*, streptococci and staphylococci.

With regard to the question of treatment, it seems that local measures cannot be entirely relied upon, for the hæmorrhage persists in spite of them. But you must make every effort to arrest the trouble, especially when umbilical bleeding is associated with hæmorrhage in various parts of the body, such as from the intestine, the pleura, the pericardium, and from the various mucous membranes. The things which have been tried, and which seem to have been successful in some cases, are the following. Ergot and ergotin should be used. If they fail you have other resources. Hall strongly recommends a quarter to half a grain of supra-renal extract, and speaks well of it. Another useful thing is a solution of gelatin, of moderate tenuity, and one to two teaspoonfuls are given every four hours. Its action is based on the well-known power of gelatin of promoting coagulation. Another suggestion which I venture to make for the treatment of these cases is, to use calcium chloride in somewhat heroic doses. In such a case I would not hesitate to give a young infant 20 to 30 gr. of calcium chloride by the mouth and repeat it every three or four hours if necessary. You should also apply calcium chloride solution locally.

So far, I have spoken of jaundice and hæmorrhage, but in treating of some other diseases of the newly-born you will note that repetition is often unavoidable; and you will see that all these diseases, or, it may be, symptoms of diseases, are interdependent. But certain causative factors stand out quite clearly, these being asphyxia, septic infection, and syphilitic infection.

We must now leave the subject of hæmorrhage and speak of a curious disease which is epidemic, such as occurred in Greystone twenty-one years ago, where I believe thirty-two children suffered. These cases were of that type known by the name of *Winckel's disease*, or as it is otherwise termed, epidemic hæmoglobinuria. It is an extremely severe and mostly a fatal disease. The cardinal symptoms of the affection are as follows. The child becomes cyanosed, and very quickly so; there is a rapid breaking up of the blood pigment, which appears in the urine. At the same time the skin becomes jaundiced, so that the combination of jaundice and cyanosis gives the child the appearance of a mulatto, a curious dusky nondescript colour. This disease comes on on the fourth or fifth day of life, and exceedingly rapidly. The child is taken very ill, and as a rule the disease does not last more than two days, in fact one may speak of it as a fulminating disease. I have mentioned the cardinal symptoms, but other symptoms are found in connection with it. One of them is a form of dysuria almost amounting to strangury; urine is passed in small quantities at frequent intervals, and micturition causes extreme pain. Coincidently with the appearances in the skin the child becomes restless, then unconscious, and finally lapses into coma. So that the affection is one which is evidently very far-reaching and very definite in its effects. Whether it is an infective disease—as is most probable, I think—or a change in the blood we do not know. But it has all the symptoms and characteristics of an acute infectious disease, especially as it is at times epidemic. The post-mortem appearances which are found in these cases are striking. Of course one naturally looks to the kidneys to ascertain what is their state. You find the kidneys large,

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dark red in colour, and when you look at microscopical sections you will find that the tubules are simply stuffed with what may be at first taken to be granules, but on examination under a high power turn out to be crystals of hæmoglobin; especially is this so in the straight tubules. The urine in the bladder is dark brown in colour and is loaded with pigment. The liver is enlarged and the spleen is found to be large, hard and red; there is a great deal of broken down blood pigment in it. So that there is evidence of the gravest disintegration of blood. I am afraid the subject is one largely of academic interest, because of the bad prognosis. So far no treatment is known for it.

There is another disease to which an observer's name is attached, which is known as *acute fatty degeneration of the newly-born*, or Buhl's disease. It is quite definite and clear in its history, is particularly prone to occur in a definite type of children. It is another example of the far-reaching effects of asphyxia neonatorum. It begins about the fifth or sixth day of life, and its course is rather slow. It may go on for some days, or it may persist for a week or two. The child who is the subject of it becomes, firstly, rather cyanotic, and then feeble, while at the same time there is some swelling of the subcutaneous tissues, which is mistaken for the next condition which I am going to mention, œdema neonatorum. But you can easily differentiate acute fatty degeneration from œdema by the fact that the skin pits on pressure. The condition of the child becomes steadily worse, until eventually it dies. The post-mortem appearances are noteworthy. They are best marked in the heart and liver and kidneys. The course of the pathological changes seems to be somewhat as follows: Firstly, there is a parenchymatous inflammation, or an increase of connective tissue, followed by an acute fatty degeneration of the essential tissue of the organ. So you find the muscular tissue of the heart replaced by stripes of fat. Also in the liver the cells are broken down and become fatty, just as they do in acute yellow atrophy. The important point about these cases, I think, is the question of diagnosis. Although it may appear to be somewhat far-fetched, I would say it is necessary you should bear in mind the results of phosphorus and arsenic poisoning. They produce similar effects, namely, acute fatty degeneration of various viscera, so that in a case which may come before your notice you should regard it as incumbent upon you to find out whether there has been any poison administered. Phosphorus poisoning is not an unlikely event, especially in the case of illegitimate children, the offspring of parents in a low class in life, who know the toxic effects of the yellow match.

The next condition I wish to speak of to-day is *œdema neonatorum*. This also, like its predecessor, occurs in children who have been the subjects of asphyxia. And if after death you examine these children you will frequently find that they are subjects of atelectasis, or imperfect expansion of the lungs. It is not uncommon in these cases, and in acute fatty degeneration, to find *post mortem* that the bases of the lungs are not expanded. Œdema neonatorum occurs frequently in weakly children, it is not so fatal as the latter condition, and the child may recover. It is seen first in the eyelids, then in the face, and it gradually extends downwards. The children who suffer from œdema neonatorum generally do very well.

Almost every newly-born child is the subject of *erythema neonatorum*. All of you must have seen examples of it. It is common to find children of a very roseate hue at birth. If the erythema deepens dermatitis follows it. The erythema is not at all significant of any serious change, in fact it is almost a normal condition, and only becomes serious when it is associated with desquamative dermatitis. The treatment is comparatively simple: to cleanse the child, rub in a simple ointment, and treat dermatitic patches with oxide of starch and zinc powder.

We must allude to the condition known as *sclerema neonatorum*, a very remarkable form of disease. It begins very often at or about the end of the first week, and is a disease which is characterised by what we may call stiffening of the subcutaneous tissue throughout the body. It frequently occurs in children who have been asphyxiated at birth, and its onset occurs with swelling and induration of the connective tissue, generally of the dorsal surfaces of the feet; thence it spreads till it reaches the trunk, and passes to the upper extremities, ultimately reaching the face. At first the curious stiffening of the subcutaneous tissue appears in patches, which gradually run together, until the whole child may appear as if he were in a plaster case, in fact there is a case recorded in which you could pick the child up by the legs and hold it out straight. The ultimate cause of death is mechanical, due to the stiffening process spreading to the deeper tissues in the mediastinum. Associated with this sclerema there is another symptom. When the disease begins, the child's temperature falls steadily, and it has been known to go as low as 84° F. At the time when these changes are going on, the skin assumes a dirty yellowish-grey tint, and there is associated with the sclerema a degree of jaundice. The disease is generally fatal, but in some cases the process has been stopped by applying artificial heat to the child. It seems that the only chance of recovery is afforded by putting the child into an incubator the moment the affection appears, and keeping it there persistently. There have been one or two recoveries recorded of cases which have been treated in this way.

January 13th, 1908.

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PNEUMONIA AND ITS COMPLICATIONS.*

By HECTOR MACKENZIE, M.D., F.R.C.P.,
Physician to St. Thomas's Hospital and to the Brompton Hospital.

AND DISCUSSION THEREON BY

Mr. A. G. R. FOULERTON.
Dr. W. P. HERRINGHAM.
Dr. W. PASTEUR.
Dr. W. J. HADLEY.
Dr. N. DALTON.
Dr. A. M. GOSSAGE.
Dr. A. C. LATHAM.
Dr. H. A. CALEY.
Dr. S. WEST.
Dr. N. I. C. TIRARD.
Dr. J. FAWCETT.
Dr. A. B. HARRIS.
And Dr. W. L. BROWN.

(*Concluded from page* 208.)

Dr. CALEY pointed out that the series of cases from St. Mary's Hospital, 437 in number, might be taken as fairly illustrative; they showed a mortality of approximately 20 per cent. The statistics as a whole seemed to have established the fact that with primary pneumonic affection of the lungs the more remote pneumococcic lesions, such as peritonitis and arthritis, were uncommon. His series tended to confirm the predominant importance of empyema, pericarditis, and heart failure. He believed Dr. Mackenzie said in his opening remarks that pericarditis was more frequent than empyema in connection with pneumonia. But in the statistics at St. Mary's Hospital extending over many years that was not the case; empyema was seen to be the more frequent complication. He had looked into the question as to the extent to which the occurrence of empyema affected the prognosis of pneumonia. The aggregate mortality for all cases was 20 per cent.; in cases complicated by empyema the mortality was 33 per cent. In order to have some check upon that he had also collected over the same period of time a series of eighty-eight consecutive cases of empyema. Most of the cases of empyema had a definite or a doubtful history of pneumonia beforehand, and the mortality of those was between 20 and 25 per cent. Therefore it seemed that the occurrence of empyema very materially increased the gravity of a case, though not necessarily to such an extent as some of the statistics suggested. Another point which seemed to be established by the figures he had looked at was that the sooner an operation for establishing drainage was carried out the better was the prognosis, both in regard to life and to the duration of the illness. Where there was a long interval from the primary pneumonia to the opening of the empyema there was a much greater likelihood of the formation of loculated collections, and the patient was consequently in greater danger. He desired specially to bring forward the question of heart failure in pneumonia. Pericarditis was only a comparatively rare cause of heart failure, but when it did occur the mortality from it was very high. In his cases pneumonia complicated by pericarditis showed a mortality of 90 per cent., and in most of those cases where careful examination was made after death there was myocarditis as well as pericarditis. Infective endocarditis accounted for only two in his series of 437 cases, though he believed Dr. Mackenzie spoke of 1–2 per cent. That seemed to be a high figure and he would be glad to know if Dr. Mackenzie was certain that no cases had been included in that list in which there had been a previous endocarditis. His own experience was that the proportion of cases of endocarditis which seemed to originate in pneumonia was smaller than Dr. Mackenzie mentioned. Whilst pericarditis and endocarditis only accounted for a very small proportion of the cases of death from heart failure, yet it was well known that death from heart failure was the usual event in the grave cases. He had therefore looked carefully into the question of the extent to which there was evidence of muscular weakness of the heart apart from the pericarditis and endocarditis. In all cases of pneumonia there was more or less myocardial weakness, and it was only when that got beyond a certain point that one could rightly regard it as a complication. He wished to raise the question as to what extent the muscular weakness of the heart in pneumonia was due to mechanical causes, and to what extent it was due to myasthenia. The dilatation of the right side of the heart was no doubt partly mechanical in origin, the outcome of the extra strain on the right ventricle caused by obstruction in the pulmonary circuit, hence the relief afforded by venesection in some cases. But that was not the only factor. Clinical and post-mortem observations showed that there might be a more general dilatation affecting the left as well as the right side of

* An address delivered before the Medical Section of the Royal Society of Medicine.

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the heart, and there might be a very marked myocardial weakness without much dilatation. On consideration it was evident that there were at least three other possible factors: toxæmia, pyrexia, and deficient oxygenation of the blood, all of which were present in grave cases of the disease. Under the heading of mechanical causes of failure of the heart, first was embarrassment of the pulmonary circulation, the second was deficient aspiration of the thorax. There were certain forms of positive pressure, intra-pleural, from fluid, and more rarely intra-pericardial from pericardial effusion. Another cause which was present in some cases was pressure from beneath the diaphragm due to dilated stomach, dilated colon, or general tympanites. Then there was the myasthenia, associated with inflammatory lesions: pericarditis, myocarditis and endocarditis. In all fatal cases the heart muscle was found to be involved more or less. The question was as to the relative importance in different cases of the mechanical and myasthenic factors, a matter of much moment in relation to treatment. Another possible factor, but which it was difficult to prove, was the toxæmic depression of the nerve centres. There were certain cases of pneumonia, especially of the fulminating type, following influenza, in which rapid death and symptoms of cardiac and respiratory failure were seen, and it was possible that some of them were due to toxæmic depression of the medullary centres. With regard to the digestive complications of pneumonia, in his series of 437 cases there were no cases of colitis, although diarrhœa was present in a certain proportion. There was one case of acute pneumonic empyema following acute colitis, and on bacteriological examination the *Bacillus coli* and the pneumococcus were both found in the pus. Dr. Pasteur had spoken of acute paralytic dilatation of the stomach. But from the point of view of prognosis Dr. Caley thought that it should be remembered that the large proportion of cases of dilated stomach were not those of the grave form, but simply due to the progressive distension caused by the gradual stretching of the walls of that organ. In regard to vaccine or serum treatment of the disease, he had knowledge of only two definite cases. One was a post-pneumococcic ulcerative endocarditis, affecting both sides of the heart, in which anti-streptococcic serum was used but without apparent effect, as the patient died. The other case was not under his own care, but was brought under his notice. The patient had an attack of pneumonia with delayed resolution, and in many respects it closely resembled tubercle, though that bacillus was not discovered by repeated examination. The pneumococcus was, however, found. An anti-pneumococcic vaccine was prepared and under its administration the patient made a good convalescence. Lastly he referred to exposure as a factor in inducing pneumonia. Dr. Latham had referred to it and adduced additional evidence to show its importance. Some years ago he (Dr. Caley) made careful inquiry into the antecedents of a large series of cases of pneumonia, and if one was careful to exclude the initial rigor as evidence of chill, that was seen to be a less common antecedent than is usually supposed, but that on the other hand in a large proportion of the cases there was evidence of antecedent nasal or naso-pharyngeal catarrh, and his own feeling was that the prime cause of the disease was exposure or other lowering factors acting in the presence of pneumococcic catarrh of the upper air passages, and not such factors alone—a consideration which had an obvious bearing in preventive measures.

Dr. Samuel West referred to the importance of the pulse-respiration ratio in the disease, though it was not pathognomonic in the sense of occurring in that disease only. That ratio was often more marked during convalescence than during the early stage of the disease, for the respirations did not fall as rapidly as the pulse. With regard to the knee-jerks in the disease they varied according to the stage of the disease. He believed that it was Dr. Stanley Barnes, of Birmingham, who made the first observations on that subject. Dr. Barnes stated, and Dr. West could confirm his observation, that in the first two or three days the knee-jerks remained normal; they then became impaired and finally disappeared, remaining absent till the eighth or ninth day, when after a short stage of increase they became normal again. Dr. Barnes also stated that the knee-jerks were important in regard to prognosis, for where they disappeared early the prognosis was less favourable, and where they disappeared late the prognosis was better He also stated that they were of use in diagnosis also, for in tubercular, non-pneumococcic pneu-

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monia they were increased, and not diminished, while in cases of septic pneumonia they were unaltered. On those points he had no personal observations to offer. Reference had also been made to the spinal symptoms, *e.g.* spasms and twitchings of muscles present in some cases of pneumonia. These were sometimes so marked as closely to simulate cerebro-spinal meningitis. These symptoms occurred only early in the disease, and were not very common. The diagnosis of pneumonia, though very puzzling at first, usually became obvious in a day or two. The last point to which he wished to refer was the question of general pneumococcic infection. A series of statistics of pneumonia had been brought forward, showing that pneumonia was followed in such and such proportions of cases by such and such kinds of infection. The figures showed that about 87 per cent. of the cases of pneumonia had no complications at all; that among the 13 per cent. of complicated cases meningitis was rare, spinal lesions rarer still, and pericarditis and peritonitis by no means common. Yet in the papers published on pneumococcal infection it was surprising to see the number of cases recorded. The arthritis following pneumonia could be taken as an illustration. The statistics showed that arthritis was a very rare affection as directly traceable to pneumonia. Yet pneumococcal arthritis seemed to be common enough without pneumonia. It used to be said that blood-infection was by no means common in pneumonia, but that was not thought now, for bacteriological investigations showed that with sufficient care the pneumococcus could be demonstrated in the blood in almost every case. From this some had been led to conclude that pneumonia was a general infection with local manifestations in the lung. The same might be said of the other pneumococcal infections. In the same way the other pneumococcal infections could be regarded as a primary blood infection, with local manifestations in the joints, peritoneum, meninges, etc. But if that were so it was curious that in the case of pneumonia these other complications were not more common, and that the other pneumococcal infections were not more frequently associated with pneumonia. One might from this conclude that the pneumococcus was a germ which was easily satisfied, and when it had produced a lesion in one part of the body it did not trouble to produce any more. That was not likely, and evidence to support such a conclusion was lacking. One was therefore driven to a different interpretation, viz. that pneumonia was a local infection, and the other infections, when they occurred, were secondary complications. It was that view towards which he thought clinical investigation and observation tended. How was it possible to reconcile those divergencies? Only it seemed by believing that the pneumococcus met with under those different conditions was not the same pneumococcus as that which was the cause of pneumonia, and he believed that further researches would lead to the same view being held in regard to the pneumococcus as were now held concerning the streptococcus. Morphological similarity did not necessarily involve pathological identity. There were now many varieties of streptococcus recognised, which, though like one another, produced different pathogenic results. In the same way research would ultimately show, he believed, that the pneumococcus of pneumonia, though resembling, was not the same as, the pneumococcus met with in many of these other infections.

Dr. TIRARD feared lest, in considering the statistics which had been brought forward, and the lessons they taught, the patient should be forgotten, and an undue amount of importance attached to the complications so as to make the physician lose heart in a serious case. He felt that particularly in regard to the importance attached by Dr. Mackenzie to pleurisy which required tapping, and with regard to gangrene and abscess of the lung. Dr. Mackenzie said that in those circumstances the disease was almost invariably fatal. That, however, was not his (Dr. Tirard's) experience. He had in his mind, although he was not prepared with statistics, many cases of each of those conditions in which the result had not been invariably fatal, nor so dangerous as one might be led to believe. With regard to nephritis, Dr. Mackenzie mentioned that a large proportion of the fatal cases between the ages of seven and nineteen had some kidney complication, and considered that complication in the kidneys to be associated with the cause of death. He (Dr. Tirard) had seen numerous cases of toxæmic symptoms in connection with the pneumonia, but he had not

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seen any cases in which one could say definitely that the symptoms were due to uræmia. On the other hand, he had seen cases in which, subsequent to the attack of acute pneumonia, permanent albuminuria resulted. He thought the large proportion of fatal cases, in which some kidney complication had been present, might have been, as suggested earlier in the debate, cases in which a pre-existent trouble had been unnoted until the time of the pneumonia. He did not think there could be associated with pneumonia acute nephritis of different characters from the ordinary nephritis, *i. e.* one which was limited to the acute stage and not likely to be followed by chronic lesions. With regard to hæmoptysis, there was no reference to that in association with pneumonia in Dr. Mackenzie's paper. He would like to know whether the people who compiled the hospital statistics regarded hæmoptysis as a necessary symptom or not. He was himself inclined to regard it as a complication, but as one which was of a hopeful nature, rather than of serious import. Sir William Jenner used to remark that in young people with severe hæmoptysis and a high temperature the prognosis was almost invariably good.

Dr. FAWCETT said that he had not much to add to the discussion, as most points had already been touched upon. He pointed out that the Guy's Hospital figures did not include "terminal" pneumonias, as Dr. Herringham had stated the St. Bartholomew's Hospital figures did. Possibly that explained the fact that the death-rate at Guy's Hospital showed a lower figure than those from the other hospitals, as when the "terminal" pneumonias were added the death-rate was brought up to about 20 per cent. In the St. Thomas's Hospital figures there was shown to have been no cases of serous effusion which required tapping, and although it was recognised that this was a somewhat rare complication, yet he thought that the St. Thomas's experience was exceptional. Therefore he investigated the fifteen cases at Guy's which had been indexed as serous effusion to see what number of them had required tapping. In only three of them had paracentesis been performed, and in each the quantity of fluid withdrawn was small, varying from half a pint to a pint. In two of the fatal cases some clear fluid was found at the autopsy; in four cases the diagnosis was arrived at from physical signs only; in two cases the diagnosis of pneumonia was doubtful, and in two more the presence of pleural effusion was equally so. In the remaining two cases a little fluid was drawn off with the exploring syringe, but the chest was not tapped. Therefore if the figures relating to cases of pleural effusion, which varied from nought to twenty-five cases, were to be any value, it was desirable that they should be analysed. Among the Guy's cases there was no case of chronic pneumonia following lobar pneumonia, and in another connection he had investigated the post-mortem records of cases of fibroid lung at Guy's Hospital for a period of twenty-four years, and there was not one certain case. There were three cases in which the condition was supposed to have followed lobar pneumonia, but on looking into the reports he thought it was doubtful if any of them had suffered from a true lobar pneumonia previously.

Dr. BUTLER HARRIS desired to refer to the vaccine treatment of pneumonia. Those who had made cultures from large quantities of blood in cases of pneumonia had invariably obtained the pneumococcus, and with that fact in mind it was difficult to understand why there were not more local manifestations. That might be owing to the very nature of the micro-organism. It was one which died very rapidly, and in fact it would only live in the most favourable surroundings. If an attempt were made to grow it outside the body it would be found to grow freely in agar which had been treated with blood serum. In several cases which had come under his notice in private practice he had practised inoculation. The first of those cases was seen with him by Dr. Hawkins. A lad, æt. 18 years, had had a definite attack of acute pneumonia at Christmas time. The left base did not wholly clear for six or seven weeks. He went on with a varying temperature and rapid loss of weight, and the clinical condition seemed to suggest acute tuberculosis. But repeated examinations of the sputum failed to reveal the tubercle bacillus. His opsonic index to tubercle was invariably normal. On the other hand, the sputum contained swarms of pneumococci, which after some difficulty he was able to grow. Dr Hawkins saw the case with him and he suggested some good might be done by a bacterial vaccine

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made from the boy's own cultures. That was done and the boy was inoculated with ten million dead pneumococci. He went through a phase which could be only likened to an ordinary crisis. There was profuse expectoration of rusty coloured material, but not purulent, and his temperature dropped and in thirty-six hours the disease was over, it having lasted seven weeks. Another patient with whom he had to deal was an alcoholic man, and he saw the patient at the seventh day of the disease. There was slight pneumonia at the left base, which had not resolved. The most remarkable feature was that he had a definite peritoneal trouble, there was effusion into his left ankle-joint and he had thrombosis of his left posterior tibial vein. He was in a state of acute delirium, but his temperature was not very high. He suggested to the man's medical attendant that it would be advisable to insert a trocar into the man's arm and run the blood into culture tubes. That was done, and by taking a large number of culture tubes it was possible to get sufficient material to make enough vaccine for a few doses within twenty-four hours. Before inoculation the man's index to the pneumococcus was ·4. Thirty-six hours after he was inoculated with ten million pneumococci the condition began to improve, *i. e.* his peritoneal trouble and the stomach and intestinal distension were better, and the effusion began to go down. Twelve hours later his temperature began to rise again and it was clear that the trouble had not been cured. The opsonic index twenty-four hours after inoculation was 1·2, but it rapidly fell away again and during the next twenty-four hours it had fallen to ·6. He was therefore repeatedly inoculated every thirty-six hours for eight days, during which time his symptoms gradually disappeared, and he eventually made a complete recovery. There was clearly in that case a general pneumococcic infection, and the opsonic indices showed that the inoculation was not merely coincident with a natural improvement in the man's condition. Recently he had had another very interesting case in which the pneumonia was not caused by the pneumococcus, but by the ordinary *Staphylococcus albus*. That patient, a girl, æt. 26 years, started with profuse hæmoptysis, which lasted two days. Her temperature then rapidly rose, and she had the physical signs of consolidation at the left apex. In addition to the signs of consolidation there were many coarse moist sounds. The examination of her sputum showed nothing but staphylococcus. Three days later the middle lobe of the right lung became involved. On the sixth day of the disease the right side of the face began to swell, and during the next two days she had a vesicular condition of her right cheek. From those vesicles a pure culture of staphylococcus was obtained. Next day there were vesicles on the right ear, and he obtained a pure culture from that. In the meantime her condition was becoming very grave and her general toxic infection seemed likely to prove fatal, her heart was failing and her temperature was 105° F., respirations 44 and pulse 104. He had made a vaccine from her staphylococcus (and it was well known that the staphylococcus grew very readily, so that it was possible in ten hours to make a vaccine from the culture). She was inoculated with a hundred million of the staphylococci and from a temperature of 104° F. the fever fell within five hours of the inoculation to 99° F., from acute delirium she sank into peaceful slumber and slept well all night. Next day her temperature rose to 101° F., but after remaining there for a few hours fell again. That night she was somewhat restless, next morning did not appear quite so well, so she was given another dose of staphylococcus vaccine. That was only forty-eight hours afterwards, at a much less interval than in the case of carbuncles and boils. Before inoculation the opsonic index was ·5, after inoculation it rose to ·9, then fell again to ·6, but four hours after the second inoculation the temperature fell and did not rise any more and the patient made an uninterrupted recovery. In the early days of anti-pneumococcic serum it failed miserably. The reason he believed was that a serum was not effective unless it was a potential vaccine, *i. e.* unless it contained the dead bodies of the infective micro-organism, and secondly because the pneumococcus was a very variable organism pathologically. Experiments in the laboratory succeeded to some extent because one strain of pneumococcus was used for the infecting animal and the same strain of serum for the inoculation. Directly variations were introduced the whole thing failed. It was necessary therefore to discard all serums, and only to employ vaccines made from the patient's particular pneumococcus.

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Dr. LANGDON BROWN said his remarks were based on those cases supplied from St. Bartholomew's Hospital in which the bacteriological evidence was complete, and, therefore, naturally most of the cases were severe and many of them fatal. With regard to the point made by Dr. West, that the pneumococcus could generally be found in the blood, and therefore how it was that metastatic deposits were so seldom found. He (Dr. Brown) found that in many cases the organism had only been made to grow by taking a comparatively large quantity of blood and inoculating it into about 150 c.c. of sterile milk, so as to dilute the bactericidal properties of the blood. So the organism could not produce metastatic deposits or toxic results until the resistance of the organism had been seriously lowered. He had tried in his series to determine whether a broad distinction could be drawn between the secondary infections due to pneumonia, and those due to other pneumococcal diseases. If the route of invasion were the ear or tonsil, thrombosis of the lateral sinus and cerebral abscess were commoner, while if the appendix or Fallopian tube were the route, peritonitis was commoner than after pneumonia. But apart from such obvious differences as these, the results of pneumococcal infection were much the same whatever the source of infection. In one case of pneumonia the fluid from the subarachnoid space was teeming with pneumococci, although there was no evidence of meningitis. This had an interesting bearing on the condition known as "cerebral pneumonia." It had been said already that the lists of figures were curtailed by the omission of cases under ten years of age. The liability to secondary pneumococcal infections was greatest in early life. The average age of death from general pneumococcal infection in his series of cases was 15½ years, *i.e.* just at that age in which the prognosis of pneumonia was particularly good. It would suggest that while the local lesion was sufficient to cause death in older persons, the more resistant young could tolerate a more extensive infection before succumbing. He would not further discuss his results as the hour was late.

Dr. HECTOR MACKENZIE, in reply, said that in reviewing the statistics supplied by the various hospitals, one was struck by the almost mathematical regularity with which the complications occurred. The same uniformity was found to exist with regard to such widely separated hospitals as those of New York and Berlin. The statistics also showed how regularly the proportion of fatal cases increased with each decade, and consequently how largely the fighting powers of the body against pneumococcus were influenced by age. It also came out clearly from the figures that sex had much to do with the incidence of pneumonia, especially among the working classes. Dr. Latham had said that the greater incidence in the male sex was largely due to exposure, and that was probably true, but he (Dr. Mackenzie) also wished to draw attention to the prevalent spitting habit among men of the working classes, as sowing the seeds of the disease. It was perhaps as well that he had not had the opportunity of seeing any of the present statistics, except those from his own hospital, St. Thomas's, when he wrote his paper. The agreement of the later figures with those he had given showed how regular were the features of the disease. The number of cases included in all the reports was 7868, and of those 1722, or 21·8 per cent., terminated in death. Taken altogether, the complications of pneumonia were uncommon, the exception rather than the rule. Empyema was the only one which could be said to be at all frequent, and that occurred in only 290 cases, or 3·7 per cent., and of these eighty-eight were fatal. The relative frequency with which empyema had occurred at the different hospitals had varied more than in the case of any other complication, and at first it did not seem quite obvious why that should be so. At Charing Cross, St. Thomas's and St. Bartholomew's, the frequency of its occurrence varied between 1·2 and 1·6 per cent., at six other hospitals between 4 per cent. and 5 per cent., while at Guy's it was 7 per cent., and at Westminster 8·2 per cent. He was surprised to find that at St. Thomas's only ten cases had been observed in the last ten years, and he thought possibly a search among the records might reveal some more. Dr. Gossage had explained in regard to Westminster Hospital that the figures included the cases of empyema admitted to the hospital with a history of previous pneumonia as well as those cases admitted for pneumonia subsequently complicated with empyema, the true percentage

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for the latter being 4·8. Simple pleural effusion was only recorded in 125 cases, or 1·6 per cent. It was remarkable that at one of the hospitals paracentesis for serous effusion was performed twenty-five times, whereas at six or seven other hospitals it seemed never to have been done at all. That was a variation in practice which he could not explain. Dr. Tirard seemed to think that a too gloomy picture had been drawn as to pneumonia and its complications. But he had been careful to point out in the paper how rare most of the complications really were. Though cases of recovery from gangrene of the lung were not unknown, he did not expect that there would have been as many as seven recoveries out of thirty-seven cases of that complication. One must still, however, regard gangrene as a very serious complication, and one in which a grave prognosis was justified, although less unfavourable than previous statistics had indicated. It was difficult to diagnose between gangrene of lung and abscess of lung with fœtor. He had stated that pericarditis was the next most common complication to empyema. In the whole series of fatal cases in the investigation, pericarditis occurred in 10·6 per cent., while in the non-fatal cases it was recorded in less than 1 per cent. In many cases pericarditis was not recognised during life, and therefore it might have been a more frequent complication among the non-fatal cases than the figures indicated. Surgical treatment was seldom called for. Dr. Fawcett and Dr. Sibley had recorded interesting cases of pyo-pericardium treated surgically, but such cases were rare. It was noteworthy that the proportion of cases of endocarditis met with at the various hospitals was nearly uniform. Altogether there were 82 cases, or just over 1 per cent., which agreed with the figures previously given. Among the fatal cases there were 58 of endocarditis, or 3·8 per cent. Only 22 cases of peritonitis had been recorded in the statistics, or ·3 per cent. of the whole. Five of those cases recovered and 17 died. Otitis and meningitis were also rare. With regard to arthritis in association with pneumonia, he thought one was apt to get an exaggerated idea of its frequency from the fact that a number of different observers had published cases, each appending references to the others which had been recorded, but altogether these did not number more than a hundred. Kidney complications seemed to be of serious import in young subjects. Under all methods of treatment pneumonia appeared to run a favourable course in the young, and most of those who died of the disease were victims of acute bacteriæmia, and it was in these cases that kidney complications occurred. There was an apparent remarkable divergence in the recorded experience of different hospitals in the matter of albuminuria. At Guy's, in 109 cases out of 727 there was albuminuria, whereas at two other hospitals only 12 cases were recorded out of 988. He thought the Guy's Hospital figures were correct, because some years ago he and others made an elaborate investigation into the complications of pneumonia, and found albuminuria present in about the same proportion of cases as the Guy's figures showed. It was not a point regarding which the compilers of the present statistics were requested to collect information, and therefore the details supplied were incomplete. Among the criticisms which he had heard, outside the room rather than in it, was, What had been the good of the discussion? Could not all that had been said be found in the text-books? His reply was that no more instructive and complete picture of the complications of pneumonia was to be found as was supplied by the collected hospital reports. Conclusions founded on the experience of one man or one hospital were apt to be narrow and erroneous, and everyone must profit by learning what was going on beyond his own limited horizon. The Society existed for the advancement of knowledge and the attainment of greater unity of opinion, and it was in regard to those diseases which were most prevalent and most deadly that it was most important that work should be done. He had been able to say little in his opening remarks about serum and vaccine treatment. Serum treatment was an acknowledged failure at the present time, but it might not long remain so. Further trials with anti-pneumonic serum with present knowledge were useless, but one could fall back on active immunisation, and the vaccine treatment of pneumonia was still practically untested. The majority of young subjects recovered, and it was proving nothing to quote cases in which they had recovered after the injection of vaccines. Therefore he advocated that the vaccine should only be used in the case of patients over twenty years of age. The results he quoted in opening were too few to be at all conclusive, but they were encouraging, and he suggested as a practical outcome of the discussion that the various hospitals should be asked to investigate the value of vaccine treatment, and that someone should be designated for each of the hospitals to carry out and supervise the necessary details in regard to vaccine treatment generally, and after a sufficient interval of trial to report as to its efficacy or otherwise.

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A SIMPLE PROCEDURE BY WHICH THE RISKS OF EXCISION OF THE RECTUM CAN BE MATERIALLY REDUCED AND THE REMOVAL OF INFECTED TISSUES RENDERED MORE COMPLETE.

MR. ARBUTHNOT LANE operated on a case illustrating a method he had adopted for some time of treating cancer of the rectum, by which he renders the removal as effectual as possible, while he reduces the risk enormously. He only applies this procedure to cases in which it is not possible to resect a portion of the bowel to establish continuity. He makes use of it in cases in which it is advisable to remove the growth and all infected tissues, and when the patient is prepared to accept the very trifling inconvenience of a colotomy in the immediate vicinity of the middle line in the substance of the rectus. The younger the patient the more necessary is it that a very radical operation should be performed.

The procedure is as follows: A vertical incision is made three quarters of an inch to the left of the middle line from the umbilicus downwards for about four inches. The incision is made in this situation as it affords easy access to the pelvis and to the sigmoid flexure, which in these cases is almost always pinned down by adhesions, just as arise in obstruction due to constipation, and because a firm scar can be obtained in which the rectus muscle controls more or less completely the exit of material from the bowel. The growth is carefully defined, together with any infected glands or mesentery.

The sigmoid is rendered mobile by the careful separation from it and from the meso-sigmoid of the acquired adhesions described in his book on 'The Operative Treatment of Constipation.' A sufficient length of this portion of the intestine having been drawn out of the abdomen, it is divided

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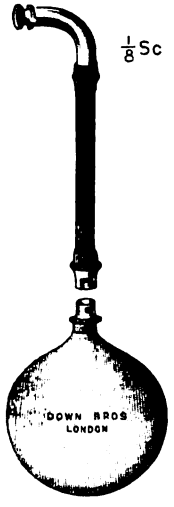
with the cautery at such a point as to leave two or three inches of the proximal bowel protruding from the wound.

The mesentery of the distal bowel is separated from the sacrum as far down as possible, and a ligature is tied firmly round the rectum at its lowest limit, all infected mesentery above the level of the ligature being thoroughly eradicated. The rectum is divided by the cautery immediately above the ligature. The ligatured stump is then prolapsed into the rectum and securely buried by purse-string and other sutures. Then the peritoneum of the pelvis lining its wall and the bladder in the male, and the uterus and broad ligament in the female, is picked up on a gut suture so that that portion of the peritoneum of the pelvis which con-



tains the stump of the rectum is completely separated from the general peritoneal cavity, and what growth still remains lies entirely below the diaphragm which has thus been constructed.

A curved tube fitted with a special rubber apparatus, which he has employed for many years, is then tied into the proximal gut, all fouling of the wound or discomfort to the patient being absolutely avoided by this means. The two or three inches of gut which projects from the wound is utilised to re-attach the glass tube as each section of bowel sloughs off with the constriction due to the ligature till the bowel is divided flush with the skin. This leaves a perfect aperture which is readily controlled by the rectus muscle and over which a cup and belt is worn without discomfort.

After the operation the patient is put upon continual saline injections subcutaneously. Two pints are introduced into the axilla at once after the operation, and during the three days following the operation about eight or ten pints are absorbed in this way by continual transfusion. To meet any risk of infection of the material introduced he devised an arrangement by means of which this can be done effectually. It has also the advantage of occupying a very small bulk in the bag of the surgeon. It is made for him by Messrs. Down Bros. It consists of a bag made of the best rubber capable of containing two pints of water. It has a metal orifice to which a connection is readily fitted. To this is joined a rubber tube to which a suitable needle can be attached. This needle is fitted with a shield which serves the double purpose of keeping it sterile and preventing any blunting of its point. A rectal nozzle is also supplied in the event of the surgeon wishing in certain cases to introduce the solution into the rectum. By using saline solution subcutaneously in this manner any sickness with the strain on the wound and the associated pain and distress is absolutely avoided, while any shock to the patient from the operation is reduced to a minimum. He first employed this continual transfusion with the most marked success in cases in which he had removed the big bowel, with the result that the risks from the operation are very much reduced, while he believes the tendency to the formation of adhesions, which is an occasional annoying sequence, is apparently obviated by it. The bag can be placed in any situation in or about the bed, or its contents can be kept warm by placing it on a hot-water bottle, etc. If the solution does not run freely pressure can be exerted in it readily. To employ it the bag is filled with water after the salt has been introduced into it. The cup, tubing, and needle are then connected, and the whole thing is put into a saucepan and boiled. The shield being taken off the needle it is introduced beneath the prepared skin and is fixed in position in a suitable dressing so as to insure asepsis at the seat of puncture. After a fortnight has elapsed the remainder of the rectum is removed from below. The rectum is separated as far as the peritoneal space below the artificial diaphragm, into which the finger enters freely, and the growth and bowel are shelled out as perfectly as possible at a mimimum of risk, since the vessels have already been ligatured and the general peritoneal cavity cannot be infected.

January 20th, 1908.

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OXALURIA, PHOSPHATURIA, AND ALBUMINURIA.*

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THE urine contains substances which have been introduced with the food (exogenous) and substances which have been formed within the body (endogenous). In some cases it is obvious which of these two sources is responsible for an abnormal constituent, in others the point is difficult to settle.

OXALURIA.

The origin of oxalates from the food is clear; their formation within the body is difficult to prove.

Small quantities of oxalates are normally present, but the only form in which they have any clinical significance is when they are deposited as calcium oxalate in envelope, or less commonly, dumb-bell crystals.

Sources of urinary oxalates. (*a*) *Direct ingestion of oxalates in the food.*—Rhubarb, spinach, strawberries, and tomatoes are the articles which, in my experience, are most prone to produce oxaluria in sufficient amount to cause symptoms. But many other articles of diet contain oxalates, *e.g.* figs, potatoes, beetroot, French beans, plums, tea, coffee, and cocoa.

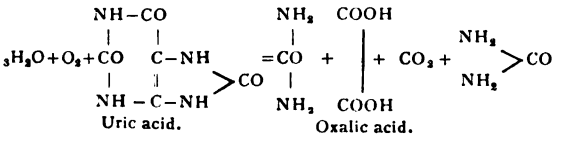
On the other hand, the following articles contain little or no oxalates: peas, asparagus, mushrooms, onions, lettuce, rice, cauliflower, pears, peaches, grapes, melons, wheat, and oats.

Baldwin's *oxalate-free diet* is composed of meat, milk, eggs, sugar, butter, wheat meal, rice, and biscuits.

The acidity of the gastric contents seems to influence the absorption of these alimentary oxalates. Thus, if there is no hydrochloric acid in the gastric juice there may be no oxalates in the urine, even if spinach is given as food; but on giving hydrochloric acid by mouth oxalates will appear. Similarly, we may reduce these alimentary oxalates by giving alkalies.

(*b*) *From gastric fermentation.* — It is curious that oxalates will appear under precisely opposite conditions—when there is no hydrochloric acid. But to produce them then, abundant fermentation must be going on, and sugar must be present, giving rise to oxalic acid by its fermentation. In this process, as the oxalates are formed in the gastric contents, it really amounts here, again, to ingestion as the source of the oxaluria. Thus Baldwin produced oxaluria in dogs by feeding on meat and excess of dextrose. A mucous gastritis with absence of free hydrochloric acid resulted, and oxalic acid was found in the gastric contents.

(*c*) *Endogenous formation.* — It is tempting to look upon uric acid as a source of oxalates in urine. This view has been widely held. In the laboratory decomposition of uric acid results in the formation of oxalic acid.



In the body a good deal of the uric acid is decomposed in the liver. If uric acid is added to an excised liver oxalic acid is formed. If the metabolism of the liver is incomplete it is easy to suppose the central carbon chain of the uric acid is not completely oxidised, when oxaluria would result.

But direct proof that this occurs is lacking, and several observers (Dunlop, Baldwin, Rendle, Short) have been unable to find any oxalates in the urine on a milk diet. We may conclude that it does not occur in sufficient quantity to produce symptoms. This clears the ground and enables us to assert that symptoms of oxaluria are the result either of ingestion of food rich in oxalates or of fermentative dyspepsia, especially with excess of carbohydrate diet.

Symptoms. (*a*) *Urinary irritation.* — Most of the cases only show this. Everyone is probably familiar with the outbreak of these cases every strawberry season, especially if the weather is very hot, so that the urine is also very concentrated. I would suggest that there are several factors at work here. Strawberries contain oxalates, cream tends to dyspepsia with reduction of hydrochloric acid, and the sugar by its fermentation produces more oxalates.

* A Lecture delivered at the Medical Graduates' College and Polyclinic.

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Micturition becomes painful and is followed by prolonged smarting. Hæmaturia is not infrequent. Envelope crystals are found abundantly in the urine. The association with intermittent albuminuria I will discuss later. I know men in whom such symptoms occur every summer, and one man in whom they occur every time he eats rhubarb.

(*b*) *Oxalate calculi* may form; they are hard mulberry stones, usually mixed with uric acid.

(*c*) *Remoter symptoms.*—It follows, from what I have said, that fermentative dyspepsia will lead to oxaluria. The so-called "remoter symptoms" and the oxaluria must be referred to a common cause. We must not regard the oxaluria as causing them.

Such symptoms often occur in crises. These crises are sometimes precipitated by worry or overwork, sometimes by some intercurrent disorder. They consist of attacks of depression and lassitude, headache, smarting pains on micturition, neuralgic pains in the back, chiefly referred to the kidney. The patient often becomes hypochondriacal. This oxaluria is the index of a very low state of health (Gee). As an example I may quote the following case:

A nervous, delicate, hard-working youth consulted me for paroxysms of pain in the left side; nothing could be made out on physical examination and a skiagram revealed nothing. The urine contained abundant crystals of calcium oxalate but no albumen. The paroxysms generally came on if he overworked or took much muscular exertion; they left him fatigued and much depressed. Subsequently he had two attacks of hæmaturia, the second rather severe and accompanied by much pain. A stone was suspected and Mr. Waring explored his kidney but found nothing to account for the symptoms. The patient still has occasional attacks of pain, but has had no more hæmaturia.

Treatment of oxaluria.—The recognised treatment is to order abstention from oxalate-containing foods and to administer magnesia, since the oxalates are more soluble in the presence of magnesia. Peas should be taken when in season, as they are poor in oxalates and rich in magnesia. We must remember that although the amount of oxalate deposited is no guide to the total oxalate excreted, it is only the deposit which causes the urinary symptoms.

The avoidance of so many things which are particularly pleasant in hot weather is an irksome prescription, and if anything can be done to mitigate the hardship it should be done.

Potassium citrate may help us; it probably acts in two ways at least:

(1) As a diuretic it dilutes the urine.

(2) By combining with the calcium it prevents the formation of calcium oxalate crystals. Martin showed that citrates threw calcium out of action by forming a soluble double salt. It is well known that citrates prevent the curdling of milk and the clotting of blood for this reason, and they have therefore been extensively used in the treatment of gastro-intestinal conditions and phlebitis, and, I think, successfully.

I have employed them in "direct" oxaluria with success also. Some observers prefer to employ lemon juice to sodium or potassium citrate, and claim that in phlebitis, at any rate, it is much more effective. Now, if we can tell our patients that they need not give up strawberries altogether if they will also drink lemon squash I do not think they have any cause for complaint, and seriously I believe we shall effect the desired object. A caution against much sugar while on this treatment should also be given.

In the nervous cases we must treat the digestive disturbance also, and direct our attention to preventing fermentation of the sugar. They generally require a holiday and change of air.

Phosphaturia.

Normally phosphates are present in the urine as—

(*a*) Acid phosphates of sodium and potassium.

(*b*) Earthy phosphates of calcium and magnesium.

It is only the latter that can form a deposit. Such deposit may occur—

(1) In the bladder, so that the last portion of the urine is milky. This is often mistaken for spermatorrhœa; the patient becomes needlessly depressed and falls an easy prey to quacks.

(2) As an iridescent pellicle on the surface of the urine when it has been passed.

(3) Only on boiling the urine, when acidification is necessary to prevent confusing it with albumen.

Calcium phosphate forms a deposit of stellar crystals, magnesium phosphate appears as rectangular plates with bevelled edges, while ammonia-

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magnesium phosphate ("triple phosphate") forms "knife-rest" or "coffin-lid" crystals.

The term "phosphaturia" is somewhat loosely applied to any condition in which these various deposits occur. But it is clear that such deposit does not necessarily mean any increase in the total output of phosphates, since under no circumstances will the sodium and potassium phosphates be precipitated, while the earthy phosphates will be deposited on adding any alkali to any urine. Quantitative estimation would be necessary to prove an increase.

Usually phosphaturia is merely a sign of diminished acidity of the urine.

Now, if there is excessive secretion of hydrochloric acid in the gastric juice there will be less acid at the disposal of the urine. Phosphaturia is therefore very common in hyperchlorhydria, and I always look for it to confirm my diagnosis of this condition.

Again, if there be cystitis the ammoniacal decomposition will lead to a deposit of phosphates which will take the form of crystals of "triple phosphates." The occurrence of "knife-rest" crystals at once suggests cystitis. Recently I saw a case where the passage of these crystals had occurred with hæmaturia at intervals for over a year. Between the attacks the urine was quite normal. But a stone was ultimately found.

There is, however, a residue of cases where the output is really increased from the normal 2½ grm. of phosphoric acid to perhaps 7 or 9 grm. Here the earthy phosphates will probably be in excess. The ratio of earthy to alkaline phosphates which is normally 1 to 2 may rise to 5 to 2. It was formerly thought that this was due to excessive breaking down of the phosphates of the brain, especially as it is common in neurasthenia, but there is no proof of this.

It must be remembered that wasting must cause an increased excretion of phosphates because the disintegration of nucleo-proteids yields phosphates. Conversely, phosphates are diminished in the urine in pregnancy and in convalescence after fevers because they are required for building up. Phosphaturia may therefore be merely a symptom of wasting. Sometimes no definite cause for the wasting can be found. Thus the editor of a "hustling" journal, æt. 38 years, consulted me for loss of weight (twenty-eight pounds in two months). He was suffering from depression and "brain-fag," but there was nothing objective except phosphaturia. Tonics and alterations in his mode of life led to great improvement. Again, a man, æt. 52 years, was sent to me to find a cause of his loss of flesh. He had formerly been in a business which had declined so much that he had recently taken up a new occupation which entailed much walking. Some swelling of the legs came on and he became very thin. Except some old adhesions round his shoulder-joint which explained the pain and limitation of movement he experienced there, I could find nothing beyond phosphaturia. As he gradually became accustomed to his new occupation he put on flesh again under ordinary tonic treatment.

Sometimes the cause of the wasting is definite. Thus recently I reported an instance of multiple myeloid tumours leading to albumosuria ('British Medical Journal,' September 14th, 1907). Here there was marked phosphaturia, no doubt in consequence of the earthy salts being set free by the destruction of the bones.

Phosphaturia may also occur in cases of marked depression without wasting. It was the only objective sign I could find in a young man who had recently gone into business on his own account, and who consulted me for paroxysms of fear of failure. It was present also in the case of a young man of the anxious nervous type, who was on the Stock Exchange and consulted me for headache, insomnia, and impairment of memory.

Probably the phosphaturia in these cases is symptomatic of a diminished formation of acid due to a general depression of metabolism.

In Ralfe's opinion cases of phosphaturia with wasting are apt to go on to serious organic diseases, if they do not rapidly yield to treatment. But I have only observed such a sequence once. A man, æt. 50 years, had much anxiety for the prolonged and ultimately fatal illness of his wife. He was much frightened because his urine had an iridescent scum on it. I found it was composed of phosphates, the urine being scarcely acid. Two or three years later he developed chronic interstitial nephritis, and had an attack of cerebral hæmorrhage which left him aphasic. I do not think, however, that there was any connection between the phosphaturia and this event. More probably the prolonged anxiety caused both depressed metabolism and high tension.

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Treatment.—We must remember that phosphaturia is a symptom and not a disease.

(1) If triple phosphates are present seek a cause for cystitis and treat that.

(2) If phosphaturia depends on hyperchlorhydria, the digestive condition requires treatment.

(3) If it is associated with wasting, it is the cause of the wasting that calls for attention.

(4) If it is a symptom of depressed metabolism, give the patient the acid he cannot make, preferably combined with tonics, and he is usually much benefited.

Now, phosphoric acid usually suits them well. If their symptoms were really due to phosphatic loss I should hardly expect that to be the case because there would already be excess of circulating phosphate, and I cannot see how the tissues can discriminate between this and the administered phosphate. Moreover, if a fish diet be given, rich in phosphates but not acid, the phosphaturia will probably be aggravated. Therefore it is simply because it is acid that phosphoric acid does good, and in my experience it does not matter whether you give phosphoric or nitro-hydrochloric acid. But give inorganic acids, and with them tincture of nux vomica.

It is an interesting point of contrast that neurasthenics tend to oxaluria with very acid urine, but to phosphaturia if the urine is not very acid.

Albuminuria.

We may classify the causes of true or renal albuminuria as follows (Tirard):

(1) Without definite structural change of renal tissue:

(*a*) Mechanical—*e.g.* from failing heart.

(*b*) Hæmatogenous—*e.g.* in the anæmias and in fevers.

(*c*) "Functional."

(2) With definite structural change of renal tissue:

"*Functional*" *albuminuria* has also been called alimentary, postural, cyclic, adolescent, to mention only a few of the names which have been given to it. Alimentary albuminuria is extremely difficult to produce. Certain experiments by D'Arcy Power upon himself are usually quoted as evidence of its existence. It is true that albumen appeared in his urine on the first day of the experiment after twelve eggs had been eaten, but it disappeared in the evening and did not reappear till the afternoon of the third day after the consumption of forty-eight eggs. It was found again during the evening of this day, after which it disappeared again and had not reappeared at the end of the experiment, by which time sixty-five eggs had been taken. Only once was the albumen present in sufficient amount to enable its coagulation point to be determined. It will be noted that there is very little connection between the number of eggs consumed and the amount of albumen. A very significant point, which seems to have escaped notice, is that at this time D'Arcy Power was aged twenty-two, and that the occurrence of the albuminuria was always observed after a considerable amount of exercise had been taken. Now, transient albuminuria after severe exercise is common in young men, as we shall see, without excess of albuminous diet.

"Functional" albuminuria is not an uncommon condition in males between puberty and marriage. Dukes found it in 16 per cent. of all the boys entering Rugby School at the ages of thirteen or fourteen. The strain of examinations seems to be a factor. A young man I knew who was working for an examination in physiology happened to test his urine. To his alarm it was loaded with albumen. He took a holiday and the urine soon became free from albumen. Some years later the albumen returned during an intercurrent illness, again disappearing when he recovered from it. He is now a healthy man in busy practice.

The subjects of this condition are usually anæmic, weedy youths with a dull, heavy aspect, and a tendency to fainting. Dukes says that boys who faint in chapel are almost certain to be albuminurics. Their hearts are often excitable, and the youths vaguely diagnosed as "weak hearts" are often examples of this condition.

Dukes divides them into three classes:

(1) By far the largest class exhibited an increased arterial tension in consequence of irritability of the vaso-motor nerves. The tension is, however, so unstable that it varies from hour to hour and day to day. This he regards as pathognomonic of the disease.

(2) The next most extensive class comprises those who have cold, clammy, congested extremities, accompanied by a large, feeble, compressible pulse arising from deficient vaso-motor control.

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(3) The remainder are the spare, highly-strung, over-sensitive neurotics.

Cold bathing may cause the albumen to appear, as may severe exercise. The proteid pressure is chiefly the serum albumen to which the kidney is more permeable. Occasionally hyaline casts may be seen, and often calcium oxalate crystals. The effect of posture is important. Albumen is absent from the urine passed first thing on rising, because this was secreted while recumbent; but it appears in urine secreted while in the upright position. With the fainting and the variable tension this strongly suggests a vaso-motor element. A lax condition of the vaso-motor system failing to compensate for the effect of gravity would allow both cerebral anæmia and back pressure on the kidney to occur.

Observations of the blood-pressure confirm this; whereas change of posture has an insignificant effect on the blood-pressure of a normal person, there may be a difference of 40 mm. between the pressure in the upright and recumbent position in these patients. I have seen a case in a girl where there was a variation of 35 mm.

Sir A. E. Wright noted a lessened coagulability of the blood in this condition which would cause diminished viscidity. Calcium salts reduce the coagulation time and increase the viscidity. He found that calcium salts would control "functional" albuminuria, whereas organic albuminuria would not be diminished and might be increased. The subjects of "functional" albuminuria have often been growing rapidly so that there is an extra demand for calcium on the part of the tissues. He therefore considered the condition "hæmatogenous" in origin and allied to a "serous exudate" such as occurs in urticaria.

These observations are very important, because, whether we accept this view of the pathology of the condition or not it provides us with a convenient clinical test for "functional" albuminuria.

Fifteen grains of calcium lactate three times a day in water should control the albumen in these cases. There is a possible fallacy, because the albumen often spontaneously disappears or is only present at certain times in the day. Systematic observations are required under hospital conditions, and these cases are, of course, commoner in private practice.

In four cases which I believed to be of this character the calcium lactate readily controlled the albumen, whereas in organic albuminuria I found it to have no effect as determined by Esbach's albuminometer.

Dr. Hingston Fox employed it in seven cases he thought were functional, and in all the albuminuria ceased, whereas in nine cases apparently organic the albuminuria persisted.

While admitting this hæmatogenous element we must not overlook the vasomotor element. I have therefore used digitalis as well as calcium lactate, and so far as I can judge the effect has been good, the general condition (such as the tendency to fainting) improving as well as the albuminuria ceasing.

The association of oxaluria and albuminuria has led to some speculation. The view commonly held is that the excretion of the calcium oxalate crystals mechanically irritates the kidney. The fact that actual hæmaturia occurs in the severer cases of oxaluria rather supports this. Berguignan regards both oxaluria and albuminuria as the result of absorption in perverted gastro-intestinal conditions, the albumen being due to direct absorption by a damaged epithelium. But a damaged epithelium takes up albumen less readily than normal, and as I shall show presently diminished protein diet is not able to diminish albuminuria. I certainly prefer the simpler hypothesis of mechanical irritation.

Prognosis.—Dr. Dukes, who has probably had a unique experience of this condition, has totally abandoned his former opinion that it tended to organic kidney disease, for he has found that his patients even thirty years later were robust men. In fact, he has only found albumen subsequently in one of these cases, and that was in a boy who had only recently left school.

The question is one of great importance in connection with life insurance. At present the attitude of the offices towards albuminuria is one of total rejection or of heavy loading. Is this fair? I believe it is justified by some, on the ground that the expectation of life is so much lower in albuminurics. Of course it is, if post-scarlatinal nephritis and other organic cases are included. They would naturally bring down the average. But has any attempt been made in such tables to exclude obviously organic cases? I believe not. Now that we have in calcium lactate

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a simply and readily applied test by which the functional can be discriminated from the organic, it seems to me that the rules of insurance companies and public services should be relaxed. It is not the duty of the medical man who examines for the companies or services to apply the test. Indeed, this would open the door to abuses, for he has no business to treat, as patients, persons coming before him for examination. But the general practitioner can fortify his patient against the ordeal of examination by administering calcium lactate, and I can see no objection to his doing so; for he will not be able to secure the acceptance of organic nephritis, but only of those who will almost certainly be free from albuminuria when adolescence is past.

Organic albuminuria.—A defective kidney not only lets things pass out which it should retain, it also retains things which it should excrete.

Many of the symptoms are due to the latter factor. Thus to take a simple example—the failure of the kidney to excrete water and salt is responsible for the œdema. In Bright's disease we have probably tended to lay too much stress on the albuminuria. In the chronic parenchymatous form no doubt the drain on the albuminous constituents may become serious—and a secondary anæmia results. But such high degrees of albuminuria are uncommon, and I think we are beginning to realise that this one symptom has unduly dominated our conception of the disease. Von Noorden thinks that any wasting is just as much explained by the monotonous diet as by the loss of albumen.

Thus when we proceed to rigidly limit the protein diet in Bright's disease are we not led away by false analogies with glycosuria? Whereas there are the following essential differences:

(1) The sugar can be replaced by other things in a diet while the protein can not.

(2) The sugar excretion is preceded by an excess of sugar in the blood; albuminuria is not preceded by excess of albumen in the blood. The latter is due to a kidney lesion, the former is not.

(3) Recent work shows that there is a great breaking down of the protein molecule into its constituent groups before it is absorbed into the body. The simple conception of Liebig according to which the protein food is simply hydrolysed into peptone and then assimilated into the tissues with no further change than dehydration is no longer held.

It has been well said that just as a gothic cathedral could not be built out of a classical temple without reducing it to its constituent stones, so the protein of the tissues cannot be built out of the protein of the food without splitting it up into its simple constituent groups.

That it may be possible for protein to "run through" the body is suggested by Power's experiments, but as it does not occur till twelve eggs are swallowed at a sitting we may say that for all practical purposes it does not occur. Even though a nephritic kidney is unduly permeable to albumen, this will not affect the question, because under ordinary circumstances the amount of albumen circulating as such is not increased by a protein meal until such enormous amounts as those just mentioned are taken.

I have tested this point several times by estimating the albuminuria on varying diets and comparing it with the urea and total nitrogen excreted.

Without burdening you with figures I may say that I added three eggs a day to the diet of patients with chronic parenchymatous nephritis without increasing the albumen in the urine. Really there is no reason why this should not be so. Formerly I used to estimate the ratio $\frac{\text{Urea N.}}{\text{Alb. N.}}$ on different diets, and if it increased when the protein of the diet was increased I concluded the increased diet was beneficial as the patient was metabolising it. But I think this position is unjustifiable and I have abandoned it. For consider what happens when we give a normal individual an excess of protein food; he turns it into urea and excretes it as quickly as possible. The mere fact that the nephritic can turn his protein into urea does not prove he has done any good with that protein. Indeed, evidence is accumulating that such protein excess never gets built up into protoplasm at all.

We know now the physiological minimum of protein is much less than the 100–125 grm. formerly ordained. Chittenden's experiments on the effect of a reduced protein diet are by now familiar to all. From observations on himself, his assistants, students, and a squad of soldiers, he concluded that weight and nitrogenous equilibrium could be kept up on 50–60 grm. protein a day or

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even less, with no diminution of physical or mental fitness, but rather an increase.

But is the physiological minimum the physiological optimum? as Hutchison pertinently asks. Experience goes to show that where we have, side by side, a race living on a protein-rich diet, and one living on a protein-poor diet, such as the Europeans and natives in India, that the incidence of an epidemic and the mortality-rate is much higher in the race that takes little protein. It is noteworthy, too, that the rapid rise of Japan corresponds fairly closely to the adoption of a more liberal nitrogenous diet.

Chittenden's work has, however, proved one fact of great importance—*i. e.* that of our protein diet very little is used for direct repair of tissue waste. Doubtless much of the rest is used as a source of energy, and it is at least probable that the ammonia groups set free from this protein excess are useful in neutralising acids that might otherwise lead to acid intoxication.

What is the bearing of all this on the dietetic treatment of chronic nephritis? We may conclude that too rigid a limitation of the protein diet with the idea of diminishing the albuminuria is bad, because it cannot effect the desired object, and deprives the patient of an essential form of nourishment. On the other hand an excessive protein diet is inadvisable, even if the patient can metabolise it, because he is getting the energy in a form that throws work on to the damaged excretory organs. What is the happy mean? I would suggest that we can arrive at it theoretically in the following way:

Chittenden's diet gives us the physiological minimum of protein. As the amount of protein in the diet has no appreciable effect on the amount of albumen in the urine, a patient with nephritis would not be able to maintain his nitrogenous equilibrium on Chittenden's diet. We must add to this diet an amount of protein equal to the albumen lost in the urine,* then we shall be giving just enough to maintain equilibrium, and yet not be taxing the kidney by calling upon it for any unnecessary work.

We should naturally avoid meat extracts and cellular organs, such as sweetbread, because they contain a large proportion of purins which, though useless for nutrition, have to be excreted by the kidney. This is contrary to the principle of physiological rest. But we must equally avoid the monotony of diet which leads to failure of appetite and consequent wasting, while it is incapable of affecting the albuminuria. We can safely permit a much greater variety of diet than is allowed on the orthodox lines. For instance, I believe from my analyses that eggs and things made with eggs certainly may be allowed.

The rules which guide us in acute nephritis or in exacerbations of chronic nephritis are somewhat different, however. "In acute affections we concentrate our attention on the diseased organ, whilst in chronic cases we keep the general condition of the patient more in view" (von Noorden). Nitrogen retention is a very prominent feature of acute nephritis, and a diet poor in nitrogen is strongly indicated. This period of retention is usually short; if it continues it is very ominous. A few days' comparative nitrogen starvation will do no harm, and may avoid grave danger.

The use of diuretics in Bright's disease.—This question is really inseparable from the consideration of the treatment of albuminuria. There has always been a tendency to regard "flushing out" the kidney as a good line of treatment in Bright's disease. But before employing it we should consider what method of diuretic we mean to employ, and how far such methods are desirable in the case before us, and how far they will achieve the end desired. Routine and indiscriminate "flushing out" is to be condemned.

Methods of producing diuresis.—The following are possible:

(1) *By vaso-dilatation in the kidney*, as by the caffeine group of drugs. These probably act as direct stimulants to the renal epithelium, the vascular change being secondary.

(2) *By vaso-constriction elsewhere*, in consequence of which the blood-pressure is raised and more blood is forced through the kidney—*e. g.* digitalis.

(3) *Increase in quantity of circulating fluid:*

* A convenient rule is this: when the reading of the albuminometer is 5 and the amount of urine is two pints, the patient is excreting as much protein as is contained in one egg. I take these figures because they admit of simple proportional calculation, and also because they represent the amount of albumen excreted in a case of chronic parenchymatous nephritis of average severity—*i. e.* 6 grm., which is the amount of protein in one egg.

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(*a*) by absorption of water from the intestine, as by giving the patient large quantities of fluid to drink; (*b*) by increasing the osmotic pressure of the blood. The saline diuretics, citrates, acetates, etc., act in this way, attracting water from the tissues into the blood-stream.

How far are these methods desirable in nephritis?

(1) Why stimulate a damaged structure? I believe I have seen caffeine, theobromine, and diuretin all produce bad effects. It is chiefly in chronic parenchymatous nephritis that one sees them employed, and there is a danger that they will cause a return of acute symptoms; hæmaturia not infrequently follows.

I have gradually come to the conclusion that this group of drugs is unsuitable for nephritis, and should be restricted to cases where diuresis is required and the kidneys are not organically diseased.

(2) Digitalis: as the blood-pressure is already raised, why raise it any further? I recently saw a case of chronic interstitial nephritis with dilating heart, which was causing a diminished urinary secretion. Digitalin injections were being given. I took the blood-pressure and found it was 200 mm. The patient's whole difficulty was that the heart could no longer work against such high blood-pressure. The effect of the treatment was to load an overworked heart still more. I suggested nitroglycerine and strophanthus instead; the pressure fell and the patient was relieved for the time. I believe the unsatisfactory results which some say digitalis gives them is due to its being employed in unsuitable cases such as this. It is, indeed, difficult to see how digitalis could be a satisfactory diuretic in cases of nephritis.

(3) In acute nephritis it is really no good to give large quantities of water with the idea of flushing the kidney, for the kidney cannot excrete it, so that it accumulates in the tissues, increasing the œdema.

This defective adjustment of the kidneys to varying water supply is an important clinical point, as the following example, quoted by Von Noorden, shows. A normal individual with an average hourly diuresis of 52 c.c. excreted an average of 723 c.c. for three hours after drinking 1800 c.c. of Salvator water; under the same circumstances a patient with acute nephritis excreting 91 c.c. hourly before, only passed 103 c.c. after. Spontaneous diuresis is the first and surest sign of convalescence.

The attempt to increase the urinary flow by increasing the osmosis into the blood is less open to objection in acute nephritis. Citrate of potassium renders the urine less acid, and, therefore, less irritating to the kidney. As the extra water is drawn from the tissues it will tend to diminish and cannot increase the œdema.

I would put it this way:

(*a*) *In acute nephritis* we cannot flush out the kidney because the inflamed organ will not respond. I believe that potassium citrate is the best drug because it does not irritate the kidney and any diuretic effect it may have is at the expense of the œdema.

(*b*) *In chronic parenchymatous nephritis* the kidney is more responsive, but it is undesirable to increase its secretion either by irritating it by caffeine and the like or by increasing the already raised pressure. Yet diuresis is here certainly desirable, because, as I have noted, the total excretion of urea follows pretty closely the excretion of water. Here, again, potassium citrate seems to be freest from objection. Another mixture I have employed, and, as far as I can judge, with success, is:

℞.	Liq. ferri acetat	♏xv
	Liq. ammon. acetat	ʒij
	Aq. camph. ad.	ʒj

(*c*) *In chronic interstitial nephritis* the kidney responds quickly to altered intake of water. But some years ago Von Noorden claimed that rather than trying to flush out the kidney it was desirable to restrict the fluids to one and a half litres a day. He maintained that this did not diminish the urea excretion while the work of the heart was spared. He considered that the polyuria was secondary to polydipsia. I have tried this plan in a good many cases since and am inclined to agree that it is preferable to the flushing-out method.

To draw the main conclusions from these somewhat discursive remarks:

Oxaluria, if the result of taking foods rich in oxalates, will probably merely cause smarting micturition, perhaps albuminuria or even hæmaturia. If the result of an intestinal intoxication it will occur in crises associated with neurasthenic and neuralgic symptoms. The former group should be treated by limiting the intake of oxalates and giving potassium citrate and magnesia, the

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latter group by treating the intestinal fermentation and limiting the intake of sugar. Formation of oxalates in the tissues has not been proved.

Phosphaturia.—If triple phosphates are present they are due to ammoniacal decomposition as in cystitis. If earthy phosphates are deposited they are usually only the result of diminished acidity, the treatment consisting of inorganic acids and nervine tonics. A genuine excess of earthy phosphates may occur as a symptom, and a relatively unimportant one, of wasting diseases.

Albuminuria.—The "functional" albuminuria of young males is largely due to a defective vasomotor control, but there is also a hæmatogenous element in it. By increasing the viscidity of the blood by calcium lactate we can control this latter factor and thereby readily distinguish the condition from organic albuminuria.

In organic albuminuria too much attention has been paid to restriction of special forms of proteid in the food, *e. g.* eggs. The diet has much less to do with the albuminuria than has been supposed. Moderation in proteid is advisable for a nephritic, because we all take more proteid than is absolutely demanded for tissue building. The excess is turned into urea and excreted, which is taxing a damaged organ unnecessarily. We can allow our chronic nephritis cases a much greater variety in diet so long as we avoid nitrogenous excess. In acute nephritis there is a tendency to nitrogenous retention, and, therefore, the proteid intake should be greatly restricted.

Diuretics are of limited application in nephritis. In no case do I advise those which act by raising the general blood-pressure or by irritating the kidney. In acute nephritis the power to excrete water is greatly diminished, despite drugs; in chronic interstitial nephritis restricted fluids are preferable, for diuresis is already excessive. It is in chronic parenchymatous nephritis that diuretics are most serviceable.

January 20th, 1908.

We have received from Burroughs Wellcome and Co. a sample of their "Soloid" Black Mercurial Lotion; it presents a convenient means of preparing a mercurial lotion, the value of which is well recognised in condylomata, in pruritus, and in many other skin affections. One, powdered and shaken with one fluid ounce of water, makes a lotion corresponding in strength to Lotio Hydrarg. Nigra. P. B. The official preparation may be more closely approximated by the addition of 24 minims of glycerin to the fluid ounce.

CASES OF CHOLECYSTECTOMY.*

By JOHN D. MALCOLM, F.R.C.S.Edin.,
Surgeon to the Samaritan Free Hospital.

I have removed the gall-bladder for the most part only when it seemed urgently necessary, and on only seven occasions. But all the patients are so well that I think I would have done better if I had performed the operation more frequently.

Case 1. My first case was that of a woman, æt. 56 years, who was sent to the Samaritan Free Hospital under my care by Dr. Tresilian, of Enfield, in 1902. The gall-bladder was full of stones and was extensively adherent, its mucous membrane being inflamed and much thickened. When the stones were removed I was unable to pass a probe into the cystic duct, and this fact, together with the unhealthy state of the mucous membrane, induced me to remove the gall-bladder. A large vein close to its base was seen and avoided, and the duct was isolated and tied, two small arteries being also ligatured. A temporary drainage tube was put in and a good recovery was made. Dr. Tresilian writes that he has been consulted very little by the patient since her operation, but she looks very well and may be considered a cure. After the bladder was removed I was still unable to introduce a probe from it into its duct, but could pass one easily from the duct to the bladder, there being a valve-like projection at the junction between the two. As the valve opened outwards drainage of the bladder in this case would have led to a free escape of bile, indicating that the ducts were patent, but if the sinus had closed symptoms of blocking of the cystic duct would have been certain to arise. The complete operation was therefore essential to success.

Case 2. My second case was that of a lady, æt. 29 years, who led a very active life and never had

* Delivered before the Surgical Section of the Royal Society of Medicine. Some of the views expressed in this paper were stated in the debate on cholecystectomy at the Exeter meeting of the British Medical Association and published in the 'Brit. Med. Journ.,' October 5th, 1907, but the paper was practically complete before that meeting, and the council of this section has kindly allowed me to re-publish these views with the cases.

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any illness except occasional indigestion pains until 1898, when she noticed a lump in her abdomen. She was never jaundiced. The doctor in attendance and a surgeon who kindly gave me these details found the lump so mobile that it evaded all attempts to locate its origin. In June, 1899, an exploratory incision showed that it was a very tense gall-bladder "shaped like a banana." The fluid and some stones were removed through an incision which was sewn up. The bladder was returned to the peritoneal cavity and the latter was closed without drainage. Except a slight swelling of the left leg there was no trouble during convalescence, and the patient left the nursing home at the end of three weeks. Afterwards she suffered much from indigestion, occasionally with fever, and I was consulted on March 8th, 1902, on account of a severe prolonged attack of pain and vomiting. The gall-bladder was then enlarged, hard, tender, and fixed to the back of a short median incision half-way between the umbilicus and the xiphoid cartilage. The temperature was steadily above 100° F. Rest and soothing treatment eased the pain without giving real relief. After consultation with Sir Douglas Powell I re-opened the abdomen on March 15th, 1902. The gall-bladder was distended by white fluid—its own secretion—which was evacuated with some half dozen stones. It was fixed in a bent position by adhesions so that its lumen was narrowed, and I could not pass my finger into its deeper parts without considerable separation of adhesions. I had told the patient that I might drain the bladder and remove it later, and I decided to adopt this course. The temperature was normal two days after the operation and convalescence was uneventful. Some more stones escaped but no bile at any time. The opening was maintained by a drainage-tube. I did not anticipate a long interval between the operations, but the patient was out of town a great deal until November, 1902, when I removed the gall-bladder. There was a calculus in the cystic duct, and it was necessary to divide the latter close to the common duct. Convalescence from this third operation was complicated by a return of the swelling of the left leg which developed after the first operation, and there was some weakness of that limb for a time, but I saw the patient recently and she was then very well. In this case the obstructing calculus was fixed between two of the folds of mucous membrane which form an imperfect spiral ridge within the cystic duct and which were unusually well developed. The stone appeared to be sufficiently loose to permit of the passage of fluid under pressure so that the contents of the bladder had slowly escaped, and the condition of prolonged tension and tenderness without any more serious development, when I first saw the patient before her second operation, was fully explained.

Case 3. The third case was that of a man, æt. 56 years, whom I was asked to see by Dr. R. D. Mackintosh, of Mortlake. The patient was a very athletic man, whose occupation necessitated daily prolonged office work. He was of very active habit and enjoyed all kinds of out-door exercise, but had suffered from indigestion for many years. In December, 1903, he had an attack of unusually severe pain, with fever, and Dr. Mackintosh diagnosed gall-stones. On the 29th of that month I removed a stone from an intensely inflamed adherent gall-bladder. The stone measured $1\frac{3}{4}$ in. by $1\frac{1}{4}$ in., and was shaped like a barrel, with one end pointed. I decided to drain for a time, and the bladder was removed on February 4th, 1904. The cystic duct was surrounded by dense adhesions and was distended almost to its junction with the common duct, so that it had to be ligatured close to the latter. The patient made a perfect recovery and has continued well. Since these notes were handed to the secretaries Dr. Mackintosh informs me that this patient, on November 2nd, was seized with severe pain over the gall-bladder region, accompanied by vomiting of a bilious matter. This continued for eight hours, and was followed by jaundice, high-coloured urine, and putty-coloured stools, which continued for four days. No stone was detected. There has been no recurrence of pain or sickness. At the time of this attack the patient was suffering from an acute nasal catarrh, and Dr. Mackintosh suggests that the illness may have been due to a catarrhal jaundice. He adds that "the general condition is splendid, the operation having cured him of persistent dyspepsia, 'sciatica' and vague lumbar pains."

Case 4. My fourth case was that of a woman, æt. 43 years, who was sent to me at the Samaritan Free Hospital by Dr. C. H. J. Watson, of Reigate.

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The history and conditions were very similar to those of Case 3. There were twenty-two stones, all measuring about half an inch in their longest axes, of an ivory-white colour, and dotted over with numerous round brown spots about two lines in diameter. One was fixed in the cystic duct. The treatment was similar to that in Case 3, and there was the same necessity for tying the cystic duct very close to the common duct. I intended to remove the bladder at the first operation in this case, but on beginning to separate it the hæmorrhage was so free that I decided to drain first. In November of this year this patient described herself as "quite well."

CASE 5. I saw the next case in consultation with Mr. J. Kingston Barton. The patient, whose age was 47, had suffered from attacks of sickness with vomiting for quite twenty years. She had a swelling over the appendix region which appeared to be cystic and somewhat mobile. There was an area of tenderness behind this swelling, and apparently separable from it. After a careful examination we came to the conclusion that the gall-bladder was enlarged and displaced downwards, and that the appendix was inflamed. Both were removed, each through its proper incision, on November 10th, 1904. The gall-bladder was of oval shape, about 4 in. in length, with very thin walls, and quite free from any adhesions. It contained two stones, but its distension was due to an elongation and narrowing, almost to obliteration, of the cystic duct. Probably the condition was congenital, and had become aggravated by the traction of the gradually enlarging bladder. The patient made a good convalescence. The attack of appendicitis was a fortunate development in this case, as it led to the removal of the gall-bladder before the changes in it gave rise to dangerous symptoms. An inflammatory attack in such a thin-walled bladder lying amongst the small intestines might have given rise to very serious complications. Mr. Barton tells me that this patient reports to him that her health was completely restored six months after the operation, that she has remained well ever since, and that there have been no more attacks of sickness.

CASE 6. The sixth case was sent to me by Dr. J. F. L. Whittingdale, of Sherborne, in 1905. I propose to publish it at greater length elsewhere. Shortly, the gall-bladder was universally adherent and a fistulous communication had formed between it and the stomach, whilst the common and hepatic ducts were dilated and contained stones. The gap in the stomach-wall was closed and the gall-bladder was drained on January 24th, 1905. The gall-bladder and the stones in the common and hepatic ducts were removed on February 28th, 1905. The patient made a good recovery and remains well.

CASE 7.—The last of these cases was that of a lady, æt. 52 years, who had jaundice at the age of five, and suffered much from pain and indigestion until she was thirteen. When her age was seventeen she had severe abdominal pain with jaundice and fever, continuing for ten weeks. Gall-stones were diagnosed, and the patient's father, who was a medical man, decided that she should be operated upon, but the pain abated, and nothing was done. After recovering from that illness the patient, who was of nervous temperament, had no symptoms that were definitely attributed to gall-stones. In 1899 an attack of appendicitis developed, and I was present when the inflamed appendix was removed. The gall-bladder was found to contain thirty-six stones from $\frac{1}{4}$ to $\frac{1}{2}$ an inch in diameter, which were removed through a second incision. The opening in the gall-bladder was sewn over with interrupted silk sutures, and it was dropped into the peritoneal cavity, which was closed without drainage. Union was immediate and recovery was good, although there were some temporary nervous manifestations, including an unexplained pain near the cicatrix, which only very gradually ceased.

Five years later the patient again complained of discomfort rather than pain in the gall-bladder region with tenderness on palpation and a slight rise of evening temperature. These evidences of mischief were persistent, although prolonged complete rest was taken. It was therefore decided to remove the gall-bladder, and this was done in the spring of 1905. There were slight adhesions, but the cystic duct was normal externally. The operation offered no difficulties; the patient made a good recovery and she remains well.

The gall-bladder showed a scar half an inch long on its mucous surface. There were three silk ligatures within it, all of which were coated with a thin layer of calculous matter. They had been

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used to close the opening in the gall-bladder at the first operation and had been discharged into it. Close to the cystic duct there was a small piece of tissue resembling granulation tissue attached to the mucous membrane, and which had probably caused a partial valve-like obstruction. The specimen is preserved in the museum of the Royal College of Surgeons (No. 2830 P. Pathological Series).

A consideration of these cases leads to the conclusion that the excision of the gall-bladder is an exceedingly satisfactory operation. One of the chief advantages of removing it as compared with its evacuation and temporary drainage might seem to be the impossibility of a further formation of gall-stones in it; but the probability of a new development of stones in this viscus after it has been cleared of them is not great. I have in my possession a letter in which the late Mr. Knowsley Thornton wrote that he had never known gall-stones to form again in the bladder after cholecystotomy. I was surprised by this assertion but I could not produce any evidence that the experience was unusual, and in agreement with it Mr. Mayo Robson has recently stated that in his practice and in that "of several surgeons whose aggregate of operations on the gall-bladder amount to fully 3000, it is universally acknowledged that the recurrence of gall-stones after cholecystotomy is an extremely rare occurrence."*

I have removed stones from the gall-bladder after it was supposed to have been cleared, but I pointed out, in 1895, that it may be difficult to be sure that stones have not been overlooked at the first operation.† I have seen only three cases in which there seemed to be little doubt that calculous matter did form in the gall-bladder after a cholecystotomy. In one instance, Case 7, related above, the pieces of silk in the bladder made the circumstances so peculiar that the case does not contradict the rule that recurrence is rare.

In another case Mr. Knowsley Thornton removed a number of stones from the gall-bladder of a lady, æt. 32 years, and with the exception of one attack in 1890 she was free from definite symptoms of gall-stones for ten years. In October, 1899, she was again seized with biliary colic, and I removed a considerable quantity of calculous matter of the consistence of partially dried putty. The stones had firm shells, but the slightest manipulation broke them in pieces so that none were removed whole. The bladder was drained, and as the ducts became clear the wound was allowed to close, and there has been no recurrence of symptoms that could be definitely attributed to gall-stones. I had an opportunity in 1904 of examining the parts in performing a hysterectomy on this patient, and found no stones. There is of course no proof that calculous matter was not left behind at the first operation in this case. The third case of reformation of gall-stones is related below.

It would seem, therefore, that the risk of a new development of these stones is not a strong argument in favour of removing the gall-bladder. But in my first case it was quite clear that a simple drainage would have been followed by symptoms of obstruction in the cystic duct, indistinguishable from those due to an impacted stone, and the following case is an example of conditions in which I would have acted more wisely in the interests of my patient if I had removed the gall-bladder. A woman, æt. 49 years, had suffered from attacks of abdominal pain, frequently accompanied by jaundice, for twenty-two years, being dangerously ill on at least two occasions. In 1901 she consulted Dr. Herbert C. Male, who brought her to me. On exposing the gall-bladder when the patient was comparatively free from pain, it was found to contain many calculi, but it was not distended. I incised the fundus and a cavity was disclosed which was occupied and filled by a single stone, measuring about half an inch in its longest diameter. I opened the main cavity of the bladder by cutting away the smaller one with the septum between the two. The deeper part was then cleared of stones and drained in the usual way. The sinus was allowed to close and the patient made a good recovery, and was free from pain about a year, but since then she has at times suffered from severe biliary colic, and has passed several stones, some quite of large size. The long history and the evident tendency to cicatricial contraction of the bladder-wall suggest that its removal would have given a better result. This operation should, I think, now be done, but the patient refuses to consider another operation. There was no obvious communication between the cavity containing the

* 'Brit. Med. Journ.,' February 4th, 1904, vol. i.

† 'Trans. Med. Soc.,' vol. xviii, p. 248.

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single stone and the rest of the gall-bladder, but Mr. W. S. Handley, who assisted me at the operation, succeeded in getting a section, showing that a sinus remained between the two.

Since I put these notes together it has been argued by Mr. Bland Sutton that in order to avoid fresh trouble it is desirable to remove the gall-bladder whenever it is interfered with surgically.* One objection to this course is that the removal is more dangerous than the drainage. This, however, is not such a strong argument as it might seem. The excision of a fairly healthy gall-bladder is, in such cases as my fifth and seventh, for example, an extremely easy and safe procedure. The increased risk is not appreciable unless the gall-bladder is much disorganised by inflammation. But it is in these cases of prolonged inflammation that a simple drainage most often fails to effect a cure, and thus when the danger of the radical operation is greater this procedure is proportionately more necessary. The increased danger is therefore not always a good argument for preserving the gall-bladder.

A better reason is that its absence does away with a convenient and safe means of draining the gall-ducts either on to the skin or into some portion of the intestine. This may become a very important consideration if an obstruction of the common duct should arise, and although it is only occasionally that the bladder would be absolutely essential as a substitute for the common duct the possibility of its being required for this purpose seems sufficiently important to make it undesirable to sacrifice the healthy gall-bladder simply because stones have formed in it, especially as a re-development of calculi is rare, and I think especially rare in a healthy gall-bladder.

In several of my cases I have drained the gall-bladder and removed it at a later date. This plan, for obvious reasons, should be restricted in its application as much as possible; but it is a well-established fact that in operating on the vermiform appendix the danger is greater if surgical interference is necessary when an acute inflammation of that part exists, and the same statement applies to operations on the gall-bladder. Moreover, in the case of the gall-ducts, the surgeon is very frequently able to give instant relief from all pain and inflammatory mischief by draining the bladder. This treatment, as a rule, is hardly accompanied by any danger, and at a subsequent date calculi in the larger ducts or the gall-bladder itself may be removed under the safest conditions.

A serious risk from interfering with an inflamed gall-bladder was strongly impressed upon me by a case of fatal hæmorrhage which I had the misfortune to meet with twelve years ago. An elderly lady, who had suffered from gall-stones for many years, was suddenly seized with intense pain in 1895, and rapidly became deeply jaundiced. She did not, as on other occasions, get relief, and after twelve days she had a rigor, with a rise of temperature to 106° F. She was then seen in consultation with her medical attendant by a physician, who recommended an immediate operation, but further consultations were desired, and I did not see the patient until there had been rigors, followed by a temperature of 106° F., or higher, on four consecutive evenings, the morning temperature being normal or subnormal. The gall-bladder, when exposed, had all the appearance of sloughed tissue. Adhesions were universal, but recent and easily separated. The bladder was distended by greenish fluid, and when it was emptied a stone was detected in the common duct, with a projection blocking the cystic duct, which was dilated so as to allow my finger to touch the stone. The latter, which appeared to be about three eighths of an inch in diameter, was easily felt with the fingers in the peritoneal cavity. As no bile was coming into the gall-bladder and none had passed into the intestine since the illness began, it seemed desirable to remove this stone. It was, therefore, broken up with the point of a knife introduced through the cystic duct and taken away piecemeal. In manipulating the parts with a view to pushing forward the stone, my fingers sank into the sloughy tissue covering it, but not so far as to touch the stone, and from the bruise thus made an uncontrollable hæmorrhage took place. It did not arise at once, but with the removal of pressure from within the gall-bladder that viscus regained its vascularity, and as I sutured it to the abdominal wall every needle puncture bled freely. It was whilst I was securing the bladder in position for drainage that the serious hæmorrhage was noticed. I divided the right rectus muscle and got a good view of the parts. There was no bleeding point, but only a general oozing from the damaged tissue. It was impossible to place a ligature round the affected area without including the hepatic and common ducts, and the hæmorrhage was too profuse to permit of the action of styptics. Pressure was ineffectual, and when I attempted to pass a ligature through the tissues every needle puncture caused more bleeding. Dr. Andrew Elliot, who was assisting me, aided by Dr. E. M. Callender, who gave the anæsthetic, very rapidly effected the infusion of saline fluid into a vein, but of course this was useless when the hæmorrhage was not arrested.

Uncontrollable hæmorrhage is described as one of the dangers of operations on jaundiced patients, and it has been attributed to a change in the blood caused by the presence of bile in it, in consequence of which coagulation is less rapid and firm. I

* 'Brit. Med. Journ.,' report of Exeter meeting of the Association, October 5th, 1907.

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have long observed that in jaundiced patients an incision through the abdominal wall is generally not accompanied by free or persistent hæmorrhage or other evidence of want of coagulability of the blood, and in the case under consideration the abdominal wall was particularly bloodless. This is the only case of fatal hæmorrhage that I have seen in any operation on the gall-ducts and the conditions seem to confirm the view that its cause is local rather than general. Moreover, John Hunter asserted that bile when mixed with the blood prevents coagulation. But he added that "we cannot suppose that in a living body it can be taken into the blood in such quantity as to produce this effect; for we find in a very severe jaundice that the blood is still capable of coagulating strongly."*

In the case I have recorded, and in a minor degree in all cases of distended and inflamed gall-bladder, there are good reasons why hæmorrhage should be a danger apart from the condition of jaundice. A focus of inflammation is always surrounded by an area of enlarged blood-vessels, and the engorged liver must offer an obstruction to the blood flow. In the case I have recorded the gall-bladder was practically bloodless from inflammation and from pressure within it. When the tension was removed the blood re-entered the wall of the gall-bladder, and the blood-vessels in it seemed to have temporarily lost their tone, and so permitted an uncontrollable loss of blood from the bruised tissues at the point of junction between the strangulated and the healthy but congested structures.

All authorities are agreed that shock leads to a distension of the large veins of the body, a condition which must also conduce to a free hæmorrhage from torn tissue in the central area of the portal system.

Hence, whilst every effort should be made to increase the coagulability of the blood before operating on a case of jaundice, a most important point in the surgery of the bile-ducts is to avoid cutting, and especially tearing or bruising, of tissues from which hæmorrhage cannot be completely controlled by ligatures, and the urgency of this is much greater when the tissues are inflamed. I attribute the hæmorrhage in the case I have recorded more to the condition of the tissues than to the condition of the blood. In this connection the necessity of seeing the parts operated upon, so strongly urged by Mr. Mayo Robson, is also important.

It seems to follow that in cases of acutely distended and inflamed gall-bladder it must be safer to be content to drain the bladder until inflammatory conditions subside, unless it is possible to pass a ligature completely beyond the diseased area. In the case I have just recorded I thought that the blocking of the cystic and common ducts necessitated the removal of the stone, but further experience has led me to believe that the relief of tension from draining the bladder would have been followed by an amelioration of symptoms and an escape of bile. The rapid return of blood to the apparently dead bladder-wall showed that there would have been no danger from gangrene. Of course, if the stone had been in the cystic duct and not too close to the common duct a ligature could easily have been applied so as to control the hæmorrhage.

Another point of practical interest in considering these cases is the frequency with which the cystic duct is dilated to, or very nearly to, its junction with the common duct, so that the surgeon must not count upon finding the inch and a half of narrow cystic duct described in anatomical works in which to choose his place for ligaturing and dividing it.

For the reasons which I have given it seems to me that the removal of the gall-bladder should be undertaken in most of the cases in which calculi have been left so long in the body that the bladder is much disorganised. There is a strong temptation to remove it in early cases also, because of the ease and safety with which this may be done and the greater rapidity of convalescence, but this procedure seems to be unnecessary in such cases, and it greatly increases the danger if drainage of the remaining ducts should be required on another occasion. Apart from the occasional extreme difficulty of the operation, this possible need for the gall-bladder to act as a drain is the only real objection to its removal in every case in which it is interfered with at all, and the ultimate decision on this question must finally rest upon the estimated danger incurred by the want of the bladder as a means of drainage, as compared with the danger of fresh mischief arising from its presence and from a second development of stones in it.

Whatever the final decision may be, the greatest necessity in connection with these cases at present is that operations should be performed earlier. In one of my cases, Case 7, the history points to the existence of calculi during more than forty years, and a record of symptoms lasting over twenty years is not uncommon. If the conditions were diagnosed and the patients would submit to operative treatment between the attacks, as in cases of appendicitis, the most urgent reasons for removing the gall-bladder would, to a very great extent, be obviated. The comparatively safe operation of evacuating and draining a healthy gall-bladder would be frequently substituted for the much more dangerous treatment of the inflamed parts, and would be practically without a death-rate, whilst those who think it right to remove the gall-bladder in all or most of these cases could do so with a very small risk.

January 20th, 1908.

* John Hunter's Works, edited by J. F. Palmer, vol. iii, 1837, p. 35.

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A CLINICAL LECTURE

ON

THE ASPIRATION OF A PORTION OF A TOOTH INTO A BRONCHUS.

By WILLIAM MURRELL, M.D., F.R.C.P.,

Physician and Lecturer on Medicine and Clinical Medicine at Westminster Hospital.

GENTLEMEN,—I beg to call your attention to a patient, a girl, æt. 22 years, who is suffering from a condition which, although not uncommon, is of sufficient interest to excite our attention. She has aspirated a portion of a tooth, a mere fragment it must be admitted, into a bronchus of the left lung. She tells us that for years she suffered from decayed teeth and that in September, 1903, she determined to have them out. Chloroform was administered, and twenty-three stumps were extracted at a sitting. For some days she was not quite herself and was confined to bed with nausea, headache, and a very distinct sense of discomfort in the mouth. On getting up at the expiration of a week she was troubled with a hard, dry, painless cough, which gradually increased in severity and ultimately became paroxysmal. Somewhat later it was accompanied by pain in the left side, intensified by taking a deep breath. The pain, from her description, seems to have been chiefly in the axillary region, but was also felt in the neighbourhood of the left mamma and the whole of the posterior portion of the chest. Her temperature for two months was persistently above normal and for some days was 105° F. The cough then altered in character and was accompanied by frothy, yellow, offensive expectoration often streaked with blood. One morning, after a restless night, she was seized with a violent paroxysm of coughing and brought up a portion of a tooth. Here is the specimen and you will observe that it is the palatine root of the first upper molar. It is considerably decayed and it is

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to be feared that it was not aseptic when aspirated. There was no abatement in the cough or expectoration, and the patient gradually lost flesh although there was no hæmoptysis or night-sweating. Obtaining no relief she came into the hospital for treatment. On admission on November 21st, 1905, she was anæmic and the cough was attended with copious expectoration, muco-purulent, not nummulated, but distinctly offensive. It varied in quantity from 2½ to 4½ ounces in the twenty-four hours; on standing it differentiated into an upper frothy portion, a middle clear stratum, and a lower opaque layer. Microscopical examination disclosed the presence of streptococci and organisms of many kinds. Although the expectoration was frequently examined no tubercle bacilli, no elastic fibres, and no crystals could be found. There was clubbing of the fingers, the appetite was poor, the tongue was clean, and there was constipation. On physical examination of the chest there was distinct diminution of expansion on the left side when viewed anteriorly, with increased vocal fremitus in the left axilla. Posteriorly the vocal fremitus was more distinct at the left than at the right base. Just internal to the inferior angle of the left scapula there was hyper-resonance with harsh inspiration. Below this the note became impaired until over the extreme left base the dulness was marked. In the eighth space inspiration was diminished, in the ninth expiration was prolonged and feeble, and in the ninth and tenth spaces voice resonance was considerably increased. The patient was radiographed but nothing was seen. There was no family history of phthisis, and the patient, although never strong, had had no previous illnesses of importance with the exception of an attack of influenza.

The temperature on admission was normal and continued normal during the whole of her stay in the hospital with the exception of a few days at long intervals, when it rose to between 102° F. and 103° F. The highest temperature noted was 104° F. and that on one occasion only.

It was obviously a case of bronchiectasis following the aspiration of the tooth. The only question was as to the mode of treatment to be adopted. Several exploratory punctures were made over the base of the left lung but nothing was obtained. An inhalation of carbolic acid and creasote was tried for some weeks without benefit. A mixture of guaiacol ♏xxiv, menthol ʒii and olive oil to ʒii was then used as an intra-tracheal injection, a drachm being introduced twice a day. This was continued for a fortnight when the patient was removed to a small ward where creasote was vaporised every morning, the doors and windows being kept closed for twenty minutes. The patient gradually lost her voice and it was found that the vocal cords were injected. This subsided and she was then ordered an inhalation of 40 per cent. formicaldehyde with five minims of the solution to be taken three times a day. These measures failing to effect any improvement formamint tablets were tried and materially lessened the amount of expectoration. At a later period tar pills, hypodermic injections of sulphate of atropine and inhalations of Izal were tried but also failed. At the end of March, 1906, there was still occasional elevation of temperature; the expectoration was from four to six ounces in the twenty-four hours, and was so fœtid that the odour was noticed at some distance from the bed. It was repeatedly examined but no tubercle bacilli, no elastic tissue and no Charcot-Leyden crystals were found. She complained of a persistently bad taste in the mouth intensified by each act of expectoration. There was a slow but persistent loss of weight. The patient left the hospital on August 7th, 1906, and returned to her home in Wales.

She was readmitted on September 20th, 1907, her condition being practically unchanged. She had gained a few pounds in weight and the temperature was normal. In addition to the previous physical signs there was distinct flattening at both apices, more marked above the right clavicle. The expectoration still measured four ounces in the twenty-four hours, was of the same character as before, not fœtid and contained no tubercle bacilli. It was felt that as a considerable number of medicinal remedies had been tried in vain no hope could be expected in that direction. The question of operation was fully discussed, but the patient was emphatic in her determination not to subject herself to any risk. Further examination of the expectoration showed that it contained streptococci in swarms, and it was accordingly determined to give her a course of anti-streptococcic injections. The Pasteur serum was administered in series of threes in the twenty-four hours, each injection consisting of 10 c.c. The first series was given on

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October 5th and was not followed by a reaction. The second series was given on the 8th, and on the 14th the temperature rose to 100° F., accompanied by headache and a papular rash on the abdomen, legs and arms. This persisted for two days and there was much itching and discomfort. The character of the expectoration was unchanged. On November 10th, the temperature having fallen to normal, a third series of 30 c.c. was given and on the following day the temperature rose to 101·2° F. and on the next day 102° F., after which it gradually subsided. During this period there was headache with loss of appetite and general malaise. The patient is disinclined to submit to further treatment and in a few days will return home.

An account of the early stages of this case was presented to the Clinical Society by Dr. F. W. Price, Medical Registrar to the Hospital.

These cases are not common but there are many on record. Amongst other foreign bodies which have been aspirated into the bronchi may be mentioned small bones, fragments of glass, plum-stones, orange-pips, pieces of walnut-shell, straws, buttons, ear-rings and toy balloons. A boy who aspirated a whistle emitted sibilant sounds with each expiration. In the case of a patient who had a tooth extracted under an anæsthetic, paroxysmal cough and dyspnœa ensued. A physician who some days later was called in in consultation, after a careful examination of the lungs gave it as his opinion that the symptoms were due to the impaction of a fragment of the tooth in a bronchus. His view was received by his colleagues with much opposition. To clear up the matter a hypodermic injection of apomorphine was given, and in the course of the vomiting which ensued something rolled out noisily into the basin. On examination it was found that it was a portion of the blade of a dental forceps, and the physician was accused of having made an incomplete diagnosis. In one case fragments of teeth were simultaneously aspirated into both lungs. The foreign substance, although usually expelled by coughing, is sometimes retained for many weeks. In these cases there is always a danger of tuberculous infection. When the symptoms are those of bronchiectasis it is well to begin with expectorants such as the carbonate and chloride of ammonium and iodide of potassium. Later on if the secretion is purulent and fœtid antiseptic inhalations such as tar, creasote, pure terebene, and formicaldehyde should be resorted to freely.

There are two points in connection with the case before us which are especially worthy of notice: (1) the nature of the anæsthetic employed, and (2) the posture of the patient during the operation. It has been ascertained that chloroform was given, and it is now generally recognised that accidents of this description are much more common with chloroform than with gas and ether. It is stated that during the operation the patient was placed flat on her back. This position is in many cases a convenient one for the dentist, but it facilitates the entrance of a foreign body into the lungs.

Although a portion of a tooth had been expelled under the circumstances described we were not unmindful that another fragment might have been retained, but although examination was directed to this point there was nothing to justify the suspicion. The question of operation was carefully considered, but the indications were not very favourable, and it was generally admitted that the result must, of necessity, be one of considerable doubt. The decided opposition both of the patient and her relatives removed the discussion from the range of practical politics. From her point of view she was probably right. She was not seriously ill, she was in no pain, and her appetite remained good; she was comfortable, she was well looked after, and probably she had no ambition and no particular object in life. An operation would have involved risk, possibly a certain amount of pain and discomfort, and undoubtedly a disturbance of the ordinary daily routine. Moreover, there was always the hope of still further improvement either as the result of the administration of antiseptics and other drugs or even spontaneously.

It was admitted, however, that the prognosis in these cases is not favourable, even apart from the incidence of phthisis. A residence in a high, dry climate might be attended with benefit, and were the patient in a better pecuniary position a winter in Egypt might be suggested. As it is, the only thing is to wait and see what time will bring forth. It is possible that a long course of cod-liver oil and iron might effect some improvement.

With the medico-legal aspects of the case I have little to say; I may mention, however, that in New York legal proceedings were taken against a dentist for negligence in allowing a fragment of a tooth to drop into the lungs during extraction. After causing paroxysmal cough for a month it was expelled. The patient was awarded $500 damages, the verdict being sustained on appeal.

January 27th, 1908.

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THREE LECTURES ON SURGICAL DISEASES OF CHILDREN.* †

By A. H. TUBBY, M.S., F.R.C.S.,
Surgeon to Westminster Hospital, and Consulting Surgeon Evelina Hospital for Sick Children.

LECTURE III.

GENTLEMEN,—On the last occasion when I lectured to you I said I would take the infective diseases of the newly-born and the affections of the umbilicus. And perhaps it will be well to begin with the septic diseases of the newly-born.

In the first place it is necessary to remember that the newly-born child is as subject to every kind of infection as is the adult, so you will find examples of smallpox, and measles, and pneumonia and other infective diseases present in them as much as in the adult. But such children are most liable to smallpox. During an epidemic of that disease I believe it is not an uncommon event for a child after birth to be found covered with the eruption of smallpox. It seems that premature children are more liable to these zymotic affections than those born at full term, and in the former they are peculiarly fatal. But apart from those diseases there are others to which the newly-born are particularly liable. Amongst these are septic disorders. The forms of septic disorder which exist in these children may be said to be three: There are ordinary septicæmia and pyæmia, tetanus neonatorum or tetanus nascentium, and there is that peculiar condition of septicæmia which is associated with Winckel's disease or acute fatty degeneration.

With regard to septicæmia and pyæmia, it is evident that there are several channels of ingress of bacteria into the body. In four-fifths of the cases it is clear that the disease is due to the entrance of organisms at the umbilicus. In the remaining one-fifth of the cases the ingress may be by means of excoriations about the mouth or genitals, or the injuries produced by forceps. The organisms which are usually found in three-fourths of the cases are the *Streptococcus* and *Staphylococcus pyogenes aureus*. You will find others. The degree of septic infection varies enormously, from a slight condition of erysipelatoid eruption to the most pronounced form of septicæmia. And the severe varieties of septicæmia are usually accompanied by conditions of gastro-enteritis, by hæmorrhage, and jaundice, as I have mentioned already. Sometimes the disease increases until the child is one mass of septic infection, showing evidences of septic peritonitis, of pleuro-pneumonia, of infarcts in the kidney, splenic affections, and lesions of the joints and bones. Such cases as this invariably die. But some severe cases after a more or less lengthened illness recover. I do not think I need say more about this, except that septic infection of this description is one of the great causes of the death of children shortly after birth. In these septic infections it is not uncommon to find a good deal of purpuric eruption.

The second group of septic infections are those known as *tetanus neonatorum* or *tetanus nascentium*. It is more common in America than in England although every now and then isolated cases occur in the dirty and crowded quarters of unwholesome towns. In America at times it is endemic, and I believe that at times it is so in the crowded cities of Eastern Europe. The cause of the condition is not far to seek. It has been ascertained by many investigations that the tetanus bacillus is constantly found in the upper layers of earth, and it is therefore easy to trace the mode of infection; especially when you remember that amongst the poorer classes a common means of dressing the umbilical cord is to put on more or less charred brown paper, which often contains tetanus bacilli. The last case but two of tetanus which I saw illustrated this point. I was called to see a little boy in the East-End who was playing with a toy pistol and shot himself in the hand three days previously. I asked where the wad was, and was told it had not been taken out. I therefore searched and found a brown paper wad. By that time he was very much advanced in tetanus, and on the fourth day, despite the use of anti-tetanic serum, he succumbed. There is no doubt that brown paper is largely made of filthy material, and

* Delivered at the Polyclinic.

† For information on many points in these lectures the lecturer is indebted to Messrs. Ashby and Wright's excellent text-book on 'Diseases of Children.'

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I think the application of brown paper to the umbilicus of the newly-born is responsible for a large number of the deaths which occur from tetanus neonatorum. The tetanus bacillus has been found at the site of section of the umbilical cord itself, and in other tissues of the body. It is possible the bacillus may gain entrance either through an abrasion of the genitals, or of the mouth, or of the skin. This disease usually appears in the child at about the time of the separation of the umbilical cord, when it begins to wither away and fall off. It often commences at the fourth day of life, and is very rarely known to appear later than the tenth, though sometimes it has been found as late as the fifteenth. There is a chronic form of the condition to which I shall allude presently. The first sign of the acute form which is noticed is a difficulty in suckling the child. This is due to the onset of trismus. There is also trouble in feeding the child with the spoon, and this increases rapidly; and at the same time the child passes into spasms; in fact he exhibits all the phenomena of tetanus such as one is accustomed to see in the adult. But the progress of this infantile tetanus is more rapid than in adolescents and adults. In fact so rapid is it that the vast majority of cases do not survive beyond forty-eight hours after the onset. Still, cases are on record in which the child has lived until the third and fourth day. A point to be remembered is that with the onset of spasm and convulsions there is a very rapid rise of temperature. The child in many cases will be found to have a temperature of 104° F., and in some of the very bad cases it will reach 106° F. or even higher. Unfortunately treatment of any kind seems to be of very little avail. The methods which have been tried have included the administration of chloral, bromides, of cannabis indica, the continuous use of chloroform, and the injection of minute doses of physostigmine, that is to say $\frac{1}{100}$th to $\frac{1}{80}$th of a grain every eight hours. Even the course of the cases which have been treated with anti-tetanic serum has seldom been retarded thereby. And when the serum was injected into the tissues around the umbilicus it was found to be unsuccessful in practically every case. Another method was tried, namely, intra-cerebral injection, but that failed to give anything like satisfactory results. So that at present the newly-born child, once it has become infected with tetanus in the acute form, is very liable to die.

The chronic form of tetanus neonatorum is not so severe. It often commences later in the child's life, but only by a few days. It runs a very tedious course, is associated with twitchings and with jaundice, and it is accompanied by a peculiar pallor. It may last for some weeks, and not infrequently the child recovers. The spasms were called by Marion Sims, trismoid, in contra-distinction to the term trismus as applied to ordinary tetanus.

With regard to the third group of septic infections, unfortunately no definite statement can be made. One can only suspect, from the peculiar changes in Winckel's disease, from the changes which occur in acute fatty degeneration, and in sclerema neonatorum, that the child is the subject of an infection of a rather mild type, and that this septic infection takes time to kill the patient by gradual alteration in the tissues of various organs. So much for the septic infections.

There is another subject to which I might take the opportunity of alluding, which is not septic but traumatic; I refer to the mastitis of babies. It is generally the result of direct irritation by ignorant midwives. You know it is the custom among the poorer classes for the midwives to pull the "breast strings" so as to extract milk from the breast. What pagan idea this is based upon—for there is no doubt it is pagan—I have no idea, but it is a practice which is unfortunately too common. The newly-born child is almost always found to have comparatively large breasts, both in boys and girls, and it contains a certain quantity of colostrum, which may persist to the third month. Of course if the breasts of the child swell on account of the large amount of milk in them, the treatment is very simple, namely to give the child a brisk purgative with a little iodide of potassium in it, and then the whole condition will subside. If the nurse or the midwife has been irritating the breast you will see not only acute irritation of the breast tissue but acute cellulitis of the parts around, which may seriously endanger life.

Now let us pass on to *diseases of the umbilicus*. They may seem to you a small thing to discuss, but they play a rather large part in the history of the infant. When spontaneous hæmorrhage was dealt with, I made rather a strong point about

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umbilical hæmorrhage, and it was described as being of two kinds: the variety in which blood issues from the larger vessels, and that which may be better described as oozing. This classification is not only useful as describing what takes place, but also from a clinical point of view. Vascular hæmorrhage, that is to say, where the bleeding occurs from the umbilical artery, is generally due to some error in properly treating the umbilical cord. A fault which is common is tying the cord with a too tight and narrow ligature, so that the coats of the arteries are torn through and hæmorrhage takes place some few hours after birth. Another fault which is not uncommon is tying the umbilical cord too short. In that case there is not sufficient room for a large clot to form. Or it may be that the cord itself is torn during parturition, and hæmorrhage takes place from a wound of the artery. But I do not think we need place much stress on traumatism, for it is purely a question of dealing with the bleeding vessel on ordinary surgical principles. But in the case of umbilical oozing the condition is very different. Twenty-four to forty-eight hours after birth oozing takes place from the umbilicus; and the striking point is that, despite every effort which is made, both local and general, to stop it, it persists. It is a fatal form of disorder, for almost all the children so afflicted die. And the children who present the symptom of oozing at the umbilicus have a definite train of symptoms; almost invariably they have jaundice, and frequently there are purpuric spots, there is definite gastro-enteritis, and all the symptoms of blood poisoning are present. In addition you find oozing taking place in children who are the subjects of congenital syphilis, or they are the children of mothers with alcoholic tendencies, or the inheritors of hæmophilia. So you must look upon this oozing as a serious matter, and as a symptom indicative of a profound disorder in the child.

We now pass on to consider *ulceration*, and by this we mean that the spot where the umbilical cord has separated does not heal properly, and a sore forms and spreads. It is usually due to want of cleanliness in treating the wound, but the ulcer heals readily as a rule if it be dressed with boracic powder and iodoform. Ulceration may be associated with the following three conditions, which are serious:

The first one is called *omphalitis*, or ulceration of the umbilicus, with inflammation of the surrounding parts. It is of two kinds. It may be associated with the formation of small abscesses and a discharge of pus, or it may present a brawny and œdematous condition of inflammation which spreads peripherally and rapidly, not associated with the formation of pus. There is, however, profound constitutional disturbance. In omphalitis the first symptom noticed is that the wound looks dusky and somewhat dirty, and there is found around the umbilicus a ring of inflamed tissue, which, as I have said, may be either red and tense, and suppurate later, or it may remain intensely brawny and œdematous. Such inflammation may spread in two directions. It may extend for a great distance around the umbilicus centripetally, but keeps well to the surface; or it may pass directly backwards and very rapidly involve the peritoneum, setting up acute peritonitis, or causing adhesion of the intestine at the umbilicus, with the subsequent formation of a fæcal fistula and death from exhaustion. The prognosis depends on the extent of the inflammation, and whether suppurating points form. It naturally depends also upon the involvement of the peritoneum. The prognosis is fair when the inflammation is not of great extent, when it keeps to the surface, and when the pus which forms can be readily evacuated. But the outlook is distinctly bad when the inflammation is of the brawny type, when it extends very widely and when it goes backwards. I have seen two cases of very severe omphalitis. One I particularly remember, and it occurred when I was clinical assistant at Guy's Hospital, in the year 1886. A small infant was brought into Clinical Ward one night the subject of gangrenous omphalitis. The abdominal wall was a mass of acute brawny inflammation. In the centre of it was a sloughy-looking place where the umbilicus should be, from which thin pus was issuing. The child was intensely ill, the legs were drawn up, there was no abdominal movement whatever, and the respiration rate, and of course the temperature also, were raised. That child died in two days. The other case which I think of at the moment was much less severe, and it occurred while I was Assistant Surgeon at the Evelina Hospital. It was a localised condition of gangrene at the umbilicus, the size of the palm of the hand. We scraped it,

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put on iodoform and boracic acid, and the child recovered. Omphalitis is an inflammation of the cellular tissue of the umbilicus, and in the cord are three very important structures: the umbilical arteries and the umbilical vein. And this remark leads me to speak of the next portion of my subject, that is to say, umbilical arteritis and umbilical phlebitis.

Umbilical arteritis does not commence in the artery itself; it commences in the surrounding tissues, probably as an ulceration or as an omphalitis. Further, it is very noteworthy that umbilical arteritis commonly occurs in infants born of mothers who become the subjects of puerperal fever, and the presumption is that the inoculation takes place from the mother by means of foul discharge of lochia, or by germs introduced at the time of parturition. The symptoms are very definite and clear, and are really an exaggeration of this condition of omphalitis to which I have referred. The trouble begins in the surrounding tissues and spreads to the arteries, thence to the blood stream. Therefore a general condition of pyæmia follows with heart symptoms, and the lung, pleura, kidney, and other viscera are affected. In some cases the viscera escape and bones and joints are involved, and metastatic abscesses form in the muscles. I do not know that any microscopical observations have been made as to the nature of the bacteria at work, but I think it probable that they will be found to be Streptococcus and *Staphylococcus pyogenes aureus*. Arteritis is more frequent in boys than in girls, and is fatal, but not so much so as the next condition I wish to describe, namely, umbilical phlebitis.

Umbilical phlebitis is inflammation of the umbilical vein, which is characterised by one striking symptom, viz. intense jaundice, of an early, striking, and persistent nature. Its onset is easily understood when you remember the course of the umbilical vein. General pyæmia follows. So far as I know, there has never been a single case of umbilical phlebitis which has recovered. The presence of jaundice distinguishes umbilical phlebitis from arteritis.

Associated with omphalitis, and with phlebitis, is the next condition I speak of, namely, *umbilical gangrene*. When umbilical gangrene is about to set in, at the site of attachment of the umbilical cord a dirty scab is often seen to form. And on it being raised there issues from beneath a small quantity of foul muddy-coloured fluid. And very quickly indeed around this protruding spot—and it often does protrude—a circle of dark angry tissue forms, which spreads until the abdominal wall is widely invaded. Not only does it spread outwards, but backwards, and many of these children, if they live long enough, become the subjects of peritonitis and fæcal fistula. It is a slower disease than umbilical arteritis and umbilical phlebitis, and seems to be associated with some other bacteria, which may account for the preponderance of gangrenous over pyæmic symptoms, as in phlebitis and arteritis. If one suspects that this condition is beginning the treatment is perfect cleanliness, scraping, and the application of peroxide of hydrogen lotion, or the use of iodoform and boracic. You must sustain the patient's strength as much as you can and give a guarded prognosis. Some recover from this condition, but very few.

So much for the septic forms of affections of the umbilicus. Those of you who have the opportunity of seeing many babies, especially at out-patients, will have cases brought to you from time to time with a little discharge from the umbilicus, or cases in which a red protruding point is seen, due to an *umbilical polypus*. When a discharge comes from the umbilicus, it is advisable to draw the parts aside, and frequently at the bottom you will see a small red prominence, which is very much like a polypus in the ear to look at. This polypus may be one of two forms: it may be composed of ordinary granulations, such as you find in the external auditory meatus, or it may be a remnant of the omphalo-mesenteric duct. In some cases if you examine it carefully with a fine probe, it is possible to find in the small polypus a minute canal. This canal on microscopic section is seen to be lined with columnar epithelium, and therefore you are dealing with the remains of a small portion of the omphalo-mesenteric duct. Simple granulations are easily treated with a little astringent or caustic. If, on the other hand, you find a small canal in the centre, it is well to ligature the polypus and await events. In almost all cases healing takes place and there is no further trouble. But occasionally there may be a persistent discharge from the umbilicus of meconium and other matters, indicating that you are dealing with a patent Meckel's diverticulum.

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I now propose to speak of *Meckel's diverticulum* and its significance. I do not think I need go very far back in embryology, but I may mention that with the appearance of the amnion, and after the separation and differentiation of the fœtal portion of the ovum from the remainder of the yolk sac, the latter is connected with the pleuro-peritoneal cavity by means of the vitelline duct. This sometimes persists and forms part of the omphalo-mesenteric duct, which ultimately develops into Meckel's diverticulum. The clinical importance of Meckel's diverticulum is very considerable. When the late Dr. Hilton Fagge wrote his text-book on medicine, he pointed out a fact in connection with intestinal obstruction, which he had verified by very careful clinical observation. He laid down a definite dictum to the following effect: that acute intestinal obstruction occurring in a young male under twenty was almost invariably due to the presence of a Meckel's diverticulum. But in view of the recent prevalence of acute appendicitis we must modify this statement. The diverticulum is more persistent in males than in females. A committee of investigation by the Anatomical Society of Great Britain and Ireland examined 769 bodies, and found 16 with Meckel's diverticulum. The length of the diverticulum varied from two centimetres to twenty-five centimetres, and from one to five centimetres in breadth. Here is another most important point, namely as to the origin of the diverticulum. It is practically always above the ileo-cæcal valve, but at different ages it is found in various positions. I will give you some of the details of those positions. Up to the sixth month of life five were examined, and the average distance of the origin of the process above the ileo-cæcal valve was twelve centimetres, or eight inches. At the ages of six months to five years there were three cases examined and the distance was thirty-two centimetres, so you notice the distance is increasing. From five to ten years there were eight cases, and the distance was forty-one centimetres. From ten to fifteen years of age there were three cases examined, and the distance was fifty centimetres. From fifteen to twenty there were eight cases, and the distance was sixty-six and a half centimetres. Over twenty years the number examined was thirty, and the distance was no less than eighty centimetres. It stands to reason that the nearer the diverticulum is to the ileo-cæcal valve and to the cæcum, the latter being fairly fixed, the less trouble it will give rise to. The omphalo-mesenteric duct, if one examines it, will be found in one of the following positions if it persists. It may be merely a fibrous cord running directly from the ileum to the umbilicus, with no canal whatever. In some cases it is open all the way, and then there is a discharge of meconium from the umbilicus and later a fæcal fistula. The next condition is that it may be partially obliterated, and the patent portion is liable to cystic dilatation. In some cases, instead of passing directly from the bowel to the umbilicus, there are processes passing off, attached to the mesentery, forming secondary bands. About ten years ago I was called one night to the hospital to a man who had had acute intestinal obstruction for three days. I opened his abdomen in the middle line and found a band passing from the umbilicus to the ilium, and underneath it there was a congested coil of intestine. The treatment was very simple, namely, to ligature the band at either end, remove it, and release the intestine. I suspected there might be other bands, and I made a long search but I found none. The band on microscopical examination was lined with columnar epithelium, and was a portion of Meckel's diverticulum. The patient recovered. But the usual experience of these cases is disappointing from the surgical point of view. You may find one band and think all is well when you have divided it, but there may be other bands which escape attention, especially as the patient is often not in a condition to stand a long operation.

Now we will consider the state of affairs in some of these cases of Meckel's diverticulum which I have mentioned. If you examine the umbilicus you will occasionally see a prolapse of mucous membrane from a Meckel's diverticulum which looks like a fleshy tumour. From the centre of this there is a small quantity of fæcal matter issuing. We have to consider what we should do by way of treatment. In many cases, where the opening is small it is sufficient to apply a little caustic or the actual cautery to it, and the opening closes up eventually. But in other cases that is not enough. Then the only thing to do is abdominal section, and remove the diverticulum. This will of course prevent future trouble. But the conditions associated with patent Meckel's

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diverticulum are not always so simple, for there are complications which occur. A prolapsed condition of the mucous membrane of Meckel's diverticulum is often associated with weakness of the abdominal wall, and you may find projecting above the Meckel's diverticulum a hernia as well, which renders the state of affairs difficult to treat. You will be careful not to ligature the hernial sac and its contents and so get into trouble.

I now pass on to say a few words about affections of the *urachus*. Possibly these may seem to you too small to take notice of, but they are, or may be, very serious. The urachus is a remnant of the allantois, and if you read the books which deal with this particular point you will see it stated that from the pleura and peritoneal cavity there sprouts out a pear-shaped process, which is the antecedent of the urachus and bladder.

The upper part of the allantois closes and becomes the superior true ligament of the bladder, and on it runs the hypogastric artery. Over it is the superior false ligament of the bladder or the layer of peritoneum which goes by that name. As to the cause of non-closure, the one usually accepted is that in fœtal life there has been a slight urethritis with obstruction, and urine collects here under pressure and persistently makes its way through the urachus and enters the amniotic cavity. There is no doubt that the urachus does contain urine, and for this reason, that distinct calculi have been found present in a patent urachus, and have been removed. The urachus may remain patent the whole way and discharge urine, or it may, instead of that, be closed at the distal and proximal ends, and fluid accumulating in it, a cyst is formed. Such cysts are situated in the median line, and are attached to the anterior abdominal wall. And when you press the abdominal wall below them you see retraction of the umbilicus at the same time. The subject of urachal cysts and fistula has been worked out with considerable care by Parteau (Thesis for the Paris M.D.). I do not think I need labour this subject of urachal cysts except to say that for such a condition there is only one treatment, and that is to operate and remove the cyst.

January 27th, 1908.

A POST-GRADUATE LECTURE

ON

THE INTESTINAL PERFORATION OF TYPHOID FEVER.

Delivered in the London School of Clinical Medicine.

By LAWRIE McGAVIN, F.R.C.S.Eng.,
Surgeon to the Dreadnought Hospital; Lecturer on Clinical Surgery and Teacher of Operative Surgery in the London School of Clinical Medicine.

GENTLEMEN,—The subject of my address to you to-day, viz. the intestinal perforation of typhoid fever, is one of special importance to physicians and surgeons alike; if, therefore, some of my opinions as a surgeon should seem to you to clash with yours in the consideration of a condition, the prophylactic treatment of which is, I admit, entirely medical, I would ask you to remember that I lay no claim to the right to teach you your own business, which is the treatment of the fever which causes the perforation. I merely propose to lay before you certain facts with regard to the surgical aspect of perforation which may serve to impress upon your mind its importance, and perhaps assist you in its diagnosis and treatment.

First of all I would have you remember that in spite of the voluminous literature on the subject already existing, our knowledge of it, at least surgically, is of comparatively recent date; and although great advances have been made in the treatment of perforation since the year 1884, when the mortality of the condition was still 100 per cent., surgery has hitherto failed to reduce this mortality below 70 to 80 per cent. You will easily understand that the difficulty of gaining individual experience of this condition must always be great when it is remembered that although typhoid fever is a common enough disease, perforation is a comparatively rare complication. Its frequency is variously estimated by Liebermeister (2000 cases) as 1·3 per cent.; by Murchison (1580 cases) as 3·5 per cent.; and by Griesinger (118 cases) as 11 per cent.; while Osler in 85 cases made it 25 per cent., but this is probably a very high estimate. Probably a fair proportion would be arrived at by taking the average of these cases, viz.

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10 per cent., but even this seems to me to be a high one.

The extreme danger of this variety of intestinal lesion may be said to be due to five factors; they are: (1) the intensely virulent nature of the intestinal contents in these cases; (2) the very common absence of preceding plastic peritonitis; (3) the markedly asthenic condition of the victims of severe typhoid fever; (4) the masking of the signs of perforation in those who are semi-comatose; (5) the delay in calling in surgical aid, which is often due to the obstinate ignorance of the relatives.

Now, in view of this latter statement, I would like you to bear in mind the startling figures given by Finney in 47 cases of perforation. Only 10 of these cases lived for more than 48 hours after the accident, while 3 died within the first 24 hours. Under these circumstances we must class typhoid perforation as a lesion whose danger and urgency are equally as great as those of perforating gastric ulcer or gangrene of the appendix; and the reason of this is quite apparent when we consider that these latter are conditions which not only are commonly heralded in by obvious premonitory signs, but which produce in their victims profound constitutional disturbance; whereas, on the other hand, the intestinal lesions of typhoid fever have already produced so grave an effect upon the patient that the significance of any slight addition to the pain, pyrexia, or pulse-rate may be quite overshadowed and the time of grace lost.

From time to time, then, you will meet with cases in which the difficulty of arriving at a conclusion as to the existence or non-existence of a perforation will tax your powers of diagnosis to the utmost, and you may be compelled to assume its occurrence rather than to diagnose it. At the same time the keenest clinicians will from the first be ever on the look-out for any premonitory sign which may point to the possible early occurrence of this dreaded accident. Some writers have held that there is no premonitory sign of any real value in these cases, but with this statement I am inclined to disagree. Those of you who have studied the matter will remember that cases were reported by Elsener and by Watson in which there was a definite localised peritonitis. These cases are sufficient to prove that perforation *is* at times preceded by peritonitis. The very slight amount, however, which is usually found points to the fact that the ulcer must approach very closely to the peritoneum before such inflammation can take place; and this is accounted for by the fact that in typhoid ulceration the actual process is more often one of slow necrosis than of acute sloughing accompanied by intense inflammation. However, the occurrence of peritonitis will in most cases be accompanied by some increase in pain and tenderness, and by a more exact localisation of these symptoms. With this will be commonly associated some definite rigidity of the lower part of the abdomen, which, although slight, will be quite obvious if it has been carefully watched for every day.

More important, perhaps, than pain is gradually increasing tenderness, especially if localised to one spot; and it is well to note that although such tenderness is usually due to the presence of commencing peritonitis, this peritonitis is not necessarily due to perforation, for in the 2000 cases analysed in Munich 2·2 per cent. were found to be suffering from peritonitis originating in the breaking down of a mesenteric gland secondary to the ulceration.

You will probably tell me that these signs are very indefinite, and provide very little on which to make a prognosis of threatening perforation, and so they are, but Nature seldom reveals her secrets to the casual observer, and close and accurate attention may be required to appreciate their presence and gauge their import. But when to these signs we add the occurrence of sharp hæmorrhage from the bowel and marked increase in the pulse-rate, it should be perfectly obvious that the ulceration is increasing in depth, and therefore you should be fully forewarned. And by this I mean, not that you should simply inform the friends that the state of the patient is one of increasing gravity—that will only frighten them and will not benefit the sufferer—I mean that you should take immediate steps to meet the probable catastrophe: first, by warning your surgeon that his services may be required at any moment; secondly, by obtaining the parents' consent to operation; and lastly, by putting in order all that may hasten matters should an operation be required. Let me urge you not to wait till perforation has actually taken place before carrying out these steps, for by so doing you may knock from under your patient the last prop on which he has

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to clean. Your surgeon may not be available at the last moment, your preparations may take some time, and the relatives are frequently dilatory and obstinate.

There is another point which is of importance as affecting the prognosis: it has been pointed out by Sydney Phillips that the abdomen of typhoid fever is seen in two different varieties, the hard or tympanitic, and the soft or doughy. It is quite clear that when a certain degree of tympanites is present the close application of the intestinal coils to each other is to some extent a safeguard, since extravasation is more likely to be limited, and perforation is somewhat less probable; tympanites must, therefore, not be regarded as altogether harmful. There is no doubt that the outlook is distinctly less favourable when perforation is met with in the second class of abdomen. Again, two types of ulcer are recognised: the acute, rapidly perforating ulcer, which in its appearance and behaviour closely resembles the acute perforating ulcer of the stomach, and the sloughy and more slowly progressive lesion which is more frequently accompanied by the premonitory signs to which I have already referred. You will sometimes find these two types of ulcer co-existing in the case, and in a small proportion of cases there may be more than one perforation; in Harte and Ashurst's 236 cases of perforation thirty-five were found to have more than one perforation—*i.e.* 14·8 per cent., and not only is this the case, but the accident of perforation may even be repeated in the same case, although this is fortunately a very rare occurrence. The time of perforation varies considerably, but it is usually said to occur at the end of the second or in the third week, the latter period being commonly that in which hæmorrhage is most likely to occur. Both of these accidents may, however, be met with much later, and at the Portland Hospital in the South African war the average date of perforation is given as the thirty-fifth day; the cases were, however, not numerous, and therefore this date may be taken as unusually late.

You will gather, then, from what I have said, that perforation is a complication to be very closely watched for; it is one in which strong suspicion may at times justifiably replace diagnosis, and it is one which we should endeavour by all means in our power to forestall.

So far we have considered the possible premonitory signs of perforation; the actual occurrence itself is usually announced by much more definite symptoms, and of these there are three which you should, I think, look upon as cardinal; they are, first, acute pain of sudden onset; secondly, vomiting with or without hiccough, usually persistent; and thirdly, an increase in the pulse-rate, which rises steadily from the first. Now, with regard to these, great stress is laid upon the first by nearly all authorities; Finney states that of 47 cases the onset was sudden in 35 and gradual in 12. Further, pain was the initial sign in 26 out of 42 cases, and was only absent in 8. The absence of pain must be looked upon as a very grave sign, and will, as a rule, only be found in those very asthenic cases in which the prognosis is already practically hopeless. There is commonly very little doubt of the presence of this pain, as in those patients still capable of expressing their feelings it is usually described as agonising abdominal cramp, and is frequently accompanied by profuse cold sweating. The vomiting and hiccough are stated to have been usually persistent in the fourteen cases referred to, and occurred at the onset only, in two cases.

But the most significant of these signs is undoubtedly the last; the pulse, which has perhaps been varying from 90 to 100 per minute, begins to rise steadily from the time of the perforation, at the rate of about 5 to 10 beats per hour, till it attains the rate of 130 to 150 per minute, and at the same time its rhythm is altered so as to assume the character known as "running." Taken in conjunction with the temperature, the pulse is perhaps the most reliable guide; the temperature, which is usually well above the normal before the occurrence of perforation, gives evidence of the catastrophe by an initial rise of two or three degrees, which is then followed by a marked drop; and from this time the significant phenomenon of a falling temperature with a rising pulse is seen, and should put you on your guard at once, even when the early sign of pain has not been present. Many observers state that they have failed to note this initial rise of temperature, and it is therefore interesting to note that it was present in Finney's cases in thirty-nine out of forty-seven, while in eight the temperature was lowered from the first.

Over and above these important signs there is a

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general increase in the abdominal symptoms; the rigidity and tenderness are now no longer localised, they are general; and the latter may assume the character of cutaneous hyperæsthesia. All these signs, however, you are well aware may be overshadowed by the general condition of the patient, who may be drowsy, apathetic, or even semi-comatose; and it is to such cases that I refer when I say that strong suspicion may at times justifiably replace diagnosis. The surgeon or physician who refuses to act—and I have known such—because he has not been able to satisfy himself that his diagnosis is correct, is likely to sacrifice his patient's life for the sake of a hide-bound principle.

There remains one aid to diagnosis which I feel sure is not sufficiently made use of in this condition, and especially in private practice, although it is, or ought to be, the regular custom in all hospitals. It is the estimation of the patient's leucocytosis, and it is, I believe, of the greatest value in these cases. In America and on the Continent repeated estimations of the patient's leucocytosis is a matter of routine practice, but in this country I fear the method is frequently omitted. Such an estimation need only be quantitative and not qualitative, and it should be the business of the practitioner to make this estimation before the arrival of his surgeon, that no time be lost. Finney gives examples of the enormous increase in the number of leucocytes in three cases of perforation, to show the value of the estimation; the numbers are remarkable. In the first there was a leucocytosis of 3800 before and of 24,000 after perforation; in the second, 6500 before and 10,600 after; and in the third, 3000 before and 16,400 after. These estimations should be undertaken in all cases of enteric fever from time to time, so that on any sudden alteration in the patient's condition a comparison can at once be drawn between the leucocytosis before and after such alteration.

The importance for long attached to the loss of liver dulness must be considered to have been much overrated, and at the present time it is certainly one of the least important signs. It must nevertheless be taken into consideration in certain cases. It is a fallacious sign, since intestines acutely distended by flatus are capable of insinuating themselves between the liver and the abdominal wall. At the same time where this sign comes on rapidly after a comparatively undistended condition of the bowel and accompanies the other signs above mentioned, it should be accorded a place in the general summary of evidence as pointing to free gas in the abdominal cavity.

I have no doubt that such reduction as has taken place in the mortality of perforation in recent years is not entirely due to the surgeon; a wider knowledge of the condition has led physicians to be more alert, and more ready to invoke the aid of the surgeon at the earliest possible moment, and in this they have very greatly contributed to the general success.

Before going into the question of operation, however, I would like to remind you of the opinions held by Van Hook with regard to this catastrophe. He pointed out that there was no rational treatment of perforation except laparotomy, and said that when perforation had occurred operation became imperative; that the only contra-indication was a moribund condition of the patient; that the stage of the fever was neither an indication for, nor a contra-indication against, operation; that the severity of the fever was not alone a contra-indication against operation; that early operation gave the best hope of recovery; that signs of peritonitis should not be waited for; that hot flushing of the abdomen was often a means of retarding collapse; and, lastly, that published statistics were strongly in favour of operation. He further gave the mortality of the operation in periods of five years as follows: From 1884 to 1888 it stood at 90 per cent.; from 1889 to 1893 at 87·5 per cent.; and from 1894 to 1898 at 72 per cent., and as far as I have been able to ascertain very little reduction has taken place since that date, the mortality still standing between 70 per cent. and 75 per cent.

Let me now trace for you briefly, gentlemen, the history of laparotomy for perforation in typhoid fever.

In 1884 the operation was first proposed by Leyden, and during this year von Mickulickz, performing a laparotomy for an undiagnosed condition, found a perforated typhoid ulcer, which he sutured; the patient recovered. In the following year, 1885, the operation was purposely but unsuccessfully undertaken by Lücke. In 1891 the operation was still unknown in this country, and no mention is made in the edition of Fagge and Pye-Smith of it. With regard to perforation this

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edition states: "This condition is obviously fatal." In 1894 Abbe published seventeen cases with three recoveries, and in 1896 a case was submitted to operation in the Sussex County Hospital, but the patient unfortunately died. In 1897 the first successful case done in this country was reported by Mr. Bowlby and Dr. (now Sir) Lauder Brunton; and Finney reported forty-seven cases with three recoveries. In 1899 Platt, of Manchester, collected 103 cases with twenty-one recoveries; and in 1901, during the Boer war, one case was operated upon in the Portland Hospital unsuccessfully. In 1903 Harte, of Philadelphia, published three successful cases. Since then many cases have been done both in this country and abroad, but the mortality remains very high.

The objections raised to operation by those who do not admit its justifiability are even more feeble than they are numerous; but as it is only fair to hear both sides of every question, we will consider them briefly. The difficulty of making a sufficiently early diagnosis is the first; that it is difficult there is no doubt, but on such dangerous ground surely the old dictum, "when in doubt, operate," was never more justified! The bad condition of the patient is another objection; but we know that that condition will assuredly grow worse if we do *not* operate, and Van Hook says that a moribund condition is the *only* contra-indication. The difficulty of finding the perforation is urged by some; there *may* be difficulty, since some perforations are very small; but surely this is one of the best arguments for operation, since if you cannot find the perforation it is your duty to drain the abdomen and chance the formation of a fistula which may be closed later. Again, the expectation of finding multiple perforations seems to frighten some people; but it must be remembered that the condition is comparatively rare: out of 236 cases of perforation collected by Harte and Ashurst, only thirty-five were multiple. To my mind the greater the damage to the intestine, the greater is it our duty to interfere without delay. We are told that in the softened condition of the bowel it is almost impossible to get sutures to hold: that sutures *do* hold, if they are properly applied, is the best answer to this; and again I would add that even if nothing else can be done there is still drainage of the abdomen. The last two objections I have heard are so foolish that I hesitate to mention them; viz. that a second perforation may occur, and that some cases have recovered without operation. Second perforations, I may say, are exceedingly rare; they can be dealt with by a second operation; no reliable proof, so far as I know, has ever been brought forward that perforation had really occurred in cases which have recovered without the performance of a laparotomy.

Let us suppose, then, that perforation has occurred, and that no delay has been incurred in providing all that is necessary for the use of the surgeon. The first question to be settled is with regard to the combating of shock. There is no possible doubt that the administration of chloroform or ether of itself contributes largely to the danger of these operations, and therefore if any other means can be found of replacing general anæsthesia half the battle will be won. For not only is there shock from a general anæsthetic, but the risk of bringing about a second perforation is favoured by the vomiting following its use. Local anæsthesia may be employed, and has been much advocated in America, but it is a clumsy and painful method, and not sufficiently rapid in its effects. Some of you, gentlemen, have seen in this hospital the method of intra-spinal analgesia applied repeatedly with success, and it is this method that I would strongly urge upon you in such cases, since not only does it provide an ideal analgesia for the purpose, but it has the immense advantage of completely abolishing shock. Even should it be determined for any reason to give a general anæsthetic, I would advise the injection of 5 cg. or 6 cg. of stovaine solution into the spinal canal for this latter reason alone.

The second step should be directed to keeping your patient warm. This may almost seem a superfluous injunction, and were it not that I frequently find it lost sight of, at least in private house operations, I would not trouble you with it. The room should be warm; a temperature of 75°–80° F. is none too great. The towels, table, blankets etc. should be well warmed and plenty of hot-water bottles available, and the bed should be thoroughly warmed *by the time the patient is returned to it:* it is useless to place the patient in a cold bed at the same time as the bottles and imagine that this represents a warm bed, and yet this is what I have repeatedly seen done, both in

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hospital and out of it. If there is no means of heating the table itself, several thicknesses of blanket, provided they are thoroughly heated through and through, will retain their heat for quite a long time.

The third step concerns the surgeon himself, viz. rapidity in operating; but you can do much to assist him in this respect by your attention to detail in providing materials such as hot water, hot lotions, hot towels, scalded basins, dishes etc. in plenty of time for the operation. Nothing is worse for the patient or more trying for the surgeon than to have to wait in the middle of the operation for more hot water to be fetched from, perhaps, the other end of the house, or to be handed two or three small and tepid napkins as an interpretation of the term "hot sterilised towels." Rapid operating must not, however, mean slovenly hurry; it would be infinitely better for the patient that the surgeon should content himself with the establishment of efficient drainage than that he should attempt the suture of a typhoid ulcer with such haste that the work is inefficiently done.

The possible sites of perforation should influence the surgeon in his choice of incision. Although the commonest site of perforation is the ileum at a point about 12 to 18 inches from the ileo-cæcal valve, perforations have occurred in the cæcum, appendix, and ascending colon: therefore all these situations can be best inspected by an incision in the region of the right semilunar line. Now as the wound must be drained the risk of post-operative hernia is considerable wherever the incision is made, but by keeping to the inner side of this line this advantage is gained, that if such a complication should arise and necessitate a second operation, the rectus muscle can, by a little adjustment, be made to cover the gap; whereas if the oblique muscles have been divided, or the incision made directly through the semilunar line, the repair of the gap by the ordinary methods is very difficult. The perforation must be sought for systematically with as little exposure of the bowels as possible, and will usually be found to be situated upon the anti-mesenteric border of the ileum. It may be a mere pinhole, or it may be a hole the size of a threepenny piece resulting from the sloughing out of one of the lymphoid patches or solitary follicles. Again, there may be, as already stated, several orifices, and accordingly the condition met with must greatly influence the treatment adopted.

A pinhole orifice may be simply inverted and rapidly closed by a double row of Lembert sutures, or by a purse-string suture such as that used for the stump of an appendicectomy. But the closure of a large perforation may considerably reduce the lumen of the bowel; this, however, is better than the risk attending even a limited enterectomy. Where the perforations are multiple and suture is impossible you will be faced by one of three alternatives: either you must establish an artificial anus and bring the damaged coil of bowel into the wound, or you must perform an enterectomy, which in such a case will perhaps be most rapidly done by closing the cut ends of the bowel and effecting a lateral anastomosis by means of a Murphy's button; or you may combine the making of an artificial anus with a lateral anastomosis, and, bringing out the damaged coil, wait for a more favourable opportunity of excising it and closing the ends. Now you will easily understand that the first of these methods is undesirable where the perforation is in the ileum and at some distance from the valve; it is likely to lead to rapid wasting and the irritation of the skin by the succus entericus. The second method is fraught with the gravest risks to the patient, first from shock, and secondly from the danger of failure to obtain primary union of the bowel in so feeble a subject. There remains, then, the third alternative, that of ileo-colic anastomosis with isolation of the damaged coil outside the abdomen, and this is, I think, the safest method under the circumstances. That there is a serious objection to the use of Murphy's button in the presence of enteritis must be admitted; the button is heavy and the bowel is soft and friable even at some distance from the seat of the disease. It should therefore not be used for end-to-end anastomosis, and only for lateral anastomosis when the patient's condition is so serious as to compel you to the most rapid method at your disposal.

Having now determined upon and carried out your method of treatment of the damaged bowel, you must turn your attention to the cleansing of the abdominal cavity. Present day teaching is somewhat at variance with itself on this question, and some surgeons hold that washing out of the abdomen is imperative, while others think it in

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judicious owing to the danger of carrying infecting material to as yet uninfected parts, or of interfering with the secretive and protective action of the peritoneum by the introduction into the abdomen of large quantities of saline. My own feeling is, I think, that of the majority of surgeons, viz. that far better results are obtained by careful sponging and mopping, and by the establishment of thorough drainage. One thing I believe all surgeons are agreed upon, that is, the great increase in mortality attendant upon evisceration for the purpose of washing the bowels.

Drainage of the abdomen to be thorough should be effected in three situations—in both loins and in the middle line. The track to be drained in either loin is that existing between the mesenteric attachment to the spine and the ascending and descending colon on either side. The pocket known as the pouch of Douglas can be drained by the median route from above the pubes, or better still, *per vaginam*, while the former route is used in the male for the recto-vesical pouch. The patient should be kept as far as possible in the semi-recumbent position in order to facilitate the gravitation of septic material to the safest position in the abdomen, viz. the pelvis, and thus protect the upper region with its dangerously sensitive organisation.

The material used for drainage is here of some practical importance, for although in ordinary cases of septic peritonitis drainage may be satisfactorily carried out by means of gauze strips, in this particular condition gauze is unsuitable; the adhesion which it acquires to the lymph-covered surface of the intestine makes its removal extremely difficult and often painful; and where the wall of the bowel, owing to the ulceration, is at certain spots only of the thickness of tissue paper, any forcible pulling on the gauze may cause a tear of the intestinal wall and result in the formation of a fæcal fistula of a very serious order. It is wiser, therefore, to use tubes of rubber or glass, and I would recommend you to employ those of moderate dimensions, viz. one half to five eighths of an inch in diameter. Through these can be passed strips of ribbon gauze, which, if not too tightly inserted, will act as efficient wicks, drawing off any accumulation of fluid without becoming adherent to the bowel. These drains, provided all goes well, should be removed on the fourth day.

Your subsequent treatment must closely follow that for general peritonitis from other causes. Continuous saline infusion should be resorted to at once, either by the subcutaneous method into the axillæ, or by the rectum; and this latter is perhaps the most satisfactory since it is free from pain. Where shock is marked you may do much good by the injection of adrenalin chloride in normal saline solution; but to be really efficacious it should be introduced directly into the veins, since if used hypodermically or by the rectum its first effect is to blanch the surrounding tissues, with the result that it does not at once pass into the circulation, and its *general* effect is greatly interfered with. I have frequently used a drachm of the solution to the pint of saline in carrying out an intra-venous injection with the best results.

Let me urge you then, gentlemen, not to be discouraged in advising operation, even when your patient's case seems almost hopeless; hopeless it will be if nothing is done for him; he may recover if only you are bold enough to act for him. And in order to show you that even the worst cases may at times react to operation, let me quote to you three cases reported by credible witnesses.

The first is one of Finney's. After operation for perforation this patient suffered two relapses of fever, otitis media, pleurisy, femoral thrombosis, and neuritis of both legs, and eventually recovered. The second is reported by Cushing, of Johns Hopkins University. In this there was a second laparotomy for suspected recurrence of perforation, and again a third for intestinal obstruction, all within one week; the patient ultimately recovered. The third is Platt's case, which underwent a relapse of three weeks after operation and recovered.

In conclusion, then, I would repeat that perforation in typhoid fever is not necessarily a fatal condition, and that it lies with you and you only to forestall the catastrophe by making all these necessary arrangements which I have mentioned, and by calling in your surgeon in sufficient time to render you the help which alone can save your patient.

January 27th, 1908.

Electric Sleep.—Professor Leduc's recent work on electric sleep has opened up a magnificent and far-reaching vista. It has given us the means of producing sensory and cerebral inhibition. In the future an intermittent current of proper frequency may be at our disposal, by means of which we can at will switch off any, or all, the nerve centres one by one. In the armamentarium of medicine there is a good array of weapons for stimulation, but comparatively few instruments for inhibition. Professor Leduc has given us a sleep-compeller, acting, not by poisoning, or counter-

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irritation, or exhaustion, but by the direct and immediate inhibition of the brain-cells themselves. The inhibition is produced by an electrical stimulation of the nerve-cells, with a rhythm which is incompatible with their physiological activity. To produce the inhibitory effect the current oscillations must be in tune with the physiological note. The frequency for the brain of the rabbit is 100 per second, and the current duration one thousandth of a second. Quietly, without a cry or movement, without the least sign of pain or discomfort, the animal sinks into a condition of deep narcosis, similar to that produced by chloroform. On the cessation of the current the animal awakens instantly, without any sign of pain or fear, or of fatigue. Professor Leduc has subjected himself to the treatment with no ill-effects, although in his case the experiment was not carried far enough to produce complete narcosis. Not only can general anæsthesia be produced, but by appropriate modifications of the process a limited local anæsthesia is also obtainable by this method. We are thus furnished with a most potent instrument for producing nervous inhibition, and we may hope ere long to see the electric sleep in use as a practical remedial agent, replacing or reinforcing the natural rest. More than this, we have some reason for supposing that each muscle and nerve is attuned to its own special period of electric stimulation, and, therefore, of electric inhibition, so that in the near future we may be able to put to sleep a tired member or an injured organ without obliterating the general consciousness. The necessity of finding some safe and simple means of inducing nerve sleep is becoming ever more important as the stress of civilisation increases, and the prevailing disease—a too widely extended and developed consciousness of our environment—becomes more acute. We may live to hear "two hours' inhibition" prescribed before an operation, or a "week's electric sleep" before an examination. The possibility of thus suspending growth and tissue changes is brought forcibly to our notice by the modern methods of forcing vegetables and flowers for the market. In the vegetable, as well as in the animal world, the axiom holds good that a period of quiescence or inhibition must precede a period of stimulated growth. The winter sleep of plants may be induced at any season of the year by placing them in cold storage, and plants whose growth has thus been retarded will develop much more rapidly on being re-awakened by warmth. More recently the ether narcosis of plants has come into use. By this method a brief nap of some couple of hours seems to fulfil all the purposes of a long winter sleep, and the plant can be forced at once into a new season of growth and activity. It would be a marvellous addition to the forces at our disposal if we had some similar means for regulating the development and activity of the animal organism, and perchance the electric sleep may enable us to procure the inhibitory rest required as a prelude to stimulated growth. At all events we may expect great developments in the electrical treatment of neurasthenia and brain affections, since Leduc has shown not only that the brain itself is within the domain of electrical treatment, but that total inhibition of the cerebral function may be produced by the mere pressure of an electric switch.

The above interesting remarks on electric sleep are taken from the first number of the first volume of the 'Proceedings of the Royal Society of Medicine' published by Longmans Green and Co, 39, Paternoster Row, London. These remarks occur in the Presidential Address delivered by Mr. W. Deane Butcher before the members of the Electro-Therapeutical Section, and anyone who approaches the thorny subject of electro-therapeutics with an open mind will agree that it is unscientific to taboo this branch of practice because it has claimed too much and been used perhaps for unworthy purposes. This first number of the 'Proceedings of the Royal Society of Medicine' commences with an address by Sir Thomas Barlow setting forth the great services done for medicine by the Clinical Society of London, now merged in the Royal Society of Medicine as its clinical section. Among the most interesting communications in this number is one by Mr. Bland-Sutton on a case of pregnancy complicated by a large cervical fibroid in which subtotal hysterectomy was performed at the fourth month; the fibroid was in the condition known as red degeneration, and the coloured illustration deserves the highest commendation; indeed, it will serve the Royal Society of Medicine as a very good example of the degree of excellence that should be sustained in illustrating their 'Proceedings.' Great praise must be accorded the first class work done in the Pathological Section under the guidance of Mr. S. G. Shattock, and, taken generally, the records of the several sections of the Society deserve more than passing notice.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON

MALIGNANT GROWTHS IN THE LONG BONES.

Delivered at St. Bartholomew's Hospital.

By ANTHONY A. BOWLBY, C.M.G., F.R.C.S., Surgeon to the Hospital.

GENTLEMEN,—There is a boy at the present time in Darker Ward whose case is in most respects a very typical one of rapidly growing periosteal sarcoma of the femur. This, very briefly, is his history: He is fifteen years of age and his residence is in Jersey. About three months ago he first noticed a small painful swelling on the inner side of his thigh, just above the right knee. For some time it was supposed to be "a sprain," as he had recently fallen on to this knee off his bicycle. The tumour, however, increased in size and in painfulness, and in three weeks from that date—a very short time—he had to keep to his bed and a doctor was sent for. The doctor, Mr. Hind, of Jersey, who in former years was house-surgeon at this hospital, diagnosed new growth. Three weeks later this growth was operated upon and removed, and a specimen was sent to London for examination. About a fortnight after the operation the tumour again made itself evident, a little lower down than before, and rapidly increased in size, and four weeks later the patient was sent to St. Bartholomew's for further advice, having been told by Mr. Hind that an amputation was the only treatment which was now available. Now we must remember that on arrival here the patient had had a long, tiring journey by boat and rail, and that he was in very great pain, for my house-surgeon, Mr. Ball, tells me that the day after admission the leg was so swollen, shiny, red and excessively painful that it almost looked as if the

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whole of the parts around the knee were in a state of acute inflammation. And to add to the inflammatory appearance the patient's temperature was 100° F. and afterwards 101° F., and his pulse 136. Subsequently the pulse-rate came down to 112. When I saw the boy a day later we found this condition: There was a very large swelling of the lower part of his thigh; he was quite unable to lift the limb off the bed, where it rested on a pillow; he could not bend the knee to the slightest extent; the swelling was most prominent at the site of the scar of the old operation, and there was slight discoloration there. When we examined the knee-joint, however, it was evident that it was not distended with fluid, and when we came to look at the whole aspect of the thigh it was clear that the tumour was entirely on the inner side of the bone. The patella could be moved without causing the patient pain, and when the thigh was sufficiently steadied there was slight movement at the knee-joint without causing pain, and there was no grating in the movement. We concluded, therefore, apart from the statement that a portion of the growth had been removed and examined, that it was a case of periosteal sarcoma of the femur. The next question to decide was, what was advisable? I advised amputation, and mainly for this reason: that the boy was in very great pain; he was indeed very ill from the pain which he was suffering and he was quite unable to move the limb. And when his father saw me and wanted to know whether amputation was going to cure the boy, I told him I was sure it would not cure him, that there was no probability of his being cured, that there was every expectation, unfortunately, that this disease would prove fatal, and that the only reason for amputating his thigh was to prevent him having any more of the pain from which he was then suffering, and to improve his condition and make him better temporarily. Then the father, naturally enough, wanted to know how long the boy was likely to live, and if I was right in the idea that the tumour was going to prove fatal, how long he would survive the amputation. I told him that the amputation was not likely to prove fatal, and the probability was that the boy would get through it very well, but that he would die probably some time within a year from growth in other parts of his body. He asked whether the tumour would come back in the limb. I said I did not think it would, that it was more likely to affect a distant part, and that as far as the tumour was concerned, when we had once cut the limb off he would probably have seen the last of it, for any recurrence would probably be in the lungs. Amputation was done, and he recovered from it very well. The temperature came down to normal at once, and it has remained at normal ever since. His pulse is quieter now than it has been since he was admitted. It is now 104, whereas when he came in it was 136. The patient altogether feels greatly relieved from the operation upon this limb, which caused him so much trouble and pain, and I think that in a short time he will be convalescent and able to leave the hospital.

I will now say a few words about these rapidly-growing periosteal sarcomata of the long bones, of which this is a characteristic and interesting example. They occur typically in patients of about this boy's age. Between ten and twenty-five is the age-period which will roughly cover the majority of such cases. You must not think, however, that they occur only between those ages, because cases have been known to occur quite late in life, and they may occur even in infancy. Here is a picture showing one of these rapidly-growing tumours involving the whole of the upper limb of a baby. Still, as I have said, the most common age at which they occur is between the tenth and the twenty-fifth years of life. The history of this boy is the history of most of these cases. With regard to the bones on which these tumours grow, the rapidity of growth in the femur is greater than in either the tibia or the fibula, and the occurrence of the growth in the femur is far more frequent than it is in the tibia, and still more than its ocucrrence in the fibula. In the bones of the upper extremity the humerus is much more often affected than either the radius or the ulna, indeed, these two long bones are comparatively seldom attacked by this particular disease. So in this boy the one bone has been attacked in which the disease is most commonly found, and it is also attacked in that part of the bone which is the most common seat of the disease. For if we were to take a large number of cases of periosteal sarcoma of the femur we should find that it was quite rare in the upper end of the bone, and rare in the shaft, though very common in the lower part, near the epiphysis.

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So you see the age of the patient, the bone which is affected and the part of the bone which is involved are all quite characteristic. The course of the case is very rapid. This tumour had been only noticed four months, and it was only three weeks from its noticeable occurrence that it was first operated upon. Yet we have it at the end of another few weeks attaining such a size and causing so much pain that the boy is glad to part with his limb. The rapidity of growth also in this case is quite typical. What more may happen to this boy I cannot tell you, but I am afraid that the rest of the history will be typical too, because most commonly these growths recur within a few months in the lungs, and they may grow in other viscera as well. Here is a very good picture showing growths studded over the lung of a girl, æt. 15 years, the same age as this boy, who was in the hospital on account of disease of the same nature—rapidly growing sarcoma of the lower part of the thigh-bone.

With regard to diagnosis, I would point out to you that in this boy there were many of the signs of inflammation, and the rapid pulse of 136 and a temperature of 101° F. seemed rather to confirm that. And in the case of the girl from whom that drawing was made I may mention that she was treated for three months under the impression that she had tubercular disease of her knee-joint. That mistake was not made in this boy, but the opportunities for making it were very great: the pain, the redness, the fever, the rapid pulse, all suggested inflammation. And I have seen yet higher temperatures than his, even as high as 103° F. in a case like this. So you must recognise that in a large number of cases of periosteal sarcoma near to a joint, and especially in these cases of periosteal sarcoma near the knee-joint, the simulation of inflammation or acute tubercular disease is very great, and it was on this account that I pointed out to you one or two reasons to indicate that this was not tubercular disease of the knee. For I told you the tumour was mainly on the inner side of the femur, that the patella was not raised, that there was no effusion in the joint, and that when the femur was steadied the tibia could be moved painlessly, and that there was no grating or roughness on movement. Therefore there was good reason for saying it was not a case of inflammation of the joint. The other difficulty that arises is that a growth of new bone in connection with a piece of retained dead bone may simulate these periosteal sarcoma exceedingly closely. Here is a specimen the tale of which is rather a tragic one. It is from a young man, æt. 21 years, and the femur, sawn longitudinally, shows on one aspect of it a large mass of soft tissue. Incisions have been made into it, and it can be seen that it is muscle infiltrated with a good deal of fibrous tissue. But it presented, clinically, as a large lump on this man's thigh, and it was on account of this that he came to the hospital some twenty years ago. The surgeon, under whose care he was, showed this man at consultations, and raised the question whether it was a case of periosteal sarcoma or whether it was a case of inflammation around some dead bone. It was decided that the right thing to do was to make an incision into this swelling and see what it was, and if it appeared to be sarcoma, that, as the disease was so high up, the only thing to do would be to amputate at his hip-joint. An incision was made, and here is the place where the knife passed down into the lump on the outer side of the thigh. A large, soft, vascular swelling was encountered, and those who saw this believed it was sarcoma, and an amputation was done. Afterwards, when the limb was examined it was found that inside this swelling, and underneath new bone, was a tiny piece of dead bone. It was really a case in which there was necrosis and death of a small piece of bone. There had been no external suppuration, no abscess, and that little piece of dead bone had been shut in by new bone, and there was all this thickening over it which simulated sarcoma. Here is another specimen which is so like an ossifying sarcoma or osteo-sarcoma of the lower end of the femur that if it had not been for the fact that there is a piece of dead bone seen on section, shut in a cavity inside the bone, you might even now mistake this specimen for one of periosteal sarcoma of the femur undergoing ossification. The question was, Had this boy a periosteal sarcoma of the lower end of his femur, or had he a piece of dead bone shut in somewhere, surrounded by a mass of new bone? As a matter of fact an incision was made here, as in the first specimen, to see whether there was or whether there was not a piece of dead bone to be discovered. It was not discovered, and the boy's thigh was amputated. And afterwards, on the

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bone being split, there in its centre was a piece of dead bone. Now, if it is possible to make an incision into a part and inspect it and yet make that mistake, it evidently must be easy in a certain number of cases to make this mistake when no incision has been made at all. But I want to point out that in the first specimen of mistaken diagnosis the swelling was in the shaft of the femur, and that is an important point to keep in mind. Periostitis resulting from a little piece of dead bone is so infinitely more common in the shaft, and periosteal sarcoma is so infinitely more common at the articular end, that you should at once be suspicious that a lump which presents itself on the shaft of the bone, at a distance from the joint, is probably not a periosteal sarcoma, although it may simulate it, and that it probably is, after all, a thickening around a sequestrum. But in addition to that, in these cases where there is new bone around a sequestrum there is, in most instances, a gradual shading off of the tumour, so that its margins are much less defined when it is an inflammatory swelling than when it is a new growth. And under these conditions you will not very often make a mistake, although I admit that sometimes, even with the greatest care, an error in diagnosis may be made. There is another condition which simulates periosteal sarcoma, and that is syphilitic disease of bone. Here I show you the two forearm bones of a man who was in the hospital with a swelling on his forearm, and he had a history of syphilis. This swelling was first believed to be syphilitic. He was treated with iodide of potassium and the arm was kept slung. But under the administration of iodide of potassium no diminution in the size of the swelling took place—in fact, it slowly increased. The patient was then shown at consultations, and the surgeons who saw him were doubtful as to the nature of the swelling, but they thought it was so like syphilis that it was worth while to continue the treatment longer. It was accordingly continued several weeks, and at the end of that time the patient was again shown at consultations, but the swelling was still larger. Under those circumstances it was decided that the best thing to do would be to put an Esmarch's bandage on the arm and cut into the swelling to see what its nature was. An incision was, therefore, made into it, it was believed to be sarcomatous, and the arm was amputated. Here are the bones macerated to show that it is a case of diffuse syphilitic osteitis and periostitis, and the soft material which simulated so closely a periosteal sarcoma as to be mistaken for it was, no doubt, gummatous material. Again, some years ago, at the Pathological Society of London, a specimen was shown of an unusual form of sarcoma of the femur. It was subsequently referred to a committee to report upon. This bone had been removed by amputation through the thigh, and when it was examined it was found that here again there had been periostitis and osteitis due to syphilis, and that as a result sarcoma had been simulated, and the man's thigh removed.

I mention these cases to illustrate to you how difficult it is sometimes to make a definite diagnosis in these cases. And you must remember another thing which the first case illustrates, and that is that gummatous material does not necessarily disappear under the administration of iodide of potassium. There is a belief, which I think is prevalent, that if you give a man iodide of potassium a swelling which is gummatous will necessarily disappear. But I can assure you that is not the fact. I think that a great number of cases of gumma undoubtedly yield to iodide of potassium, and that the majority are probably immediately diminished by its administration, but there are others which do not yield to it, and some of them will completely yield to small doses of mercury. Therefore, I say, do not pin your faith upon the administration of iodide of potassium and its results, when you are administering it with the expectation that a swelling of a bone will be made to disappear. But if you do think it is syphilis do not hesitate to give the patient mercury as well as iodide of potassium, for although there is rather a fashion to give mercury in the early stages of syphilis and not in the later stages, I think it is a good thing to give mercury in any stage of syphilis.

I now want to point out something else to you in connection with this specimen on the table. Here is a section of the femur of the boy whose case I began by alluding to, the patient whose thigh was amputated last week. You will see in the first place that there is, by the side of the femur, a whitish growth. If you look at the other side you will see there is a large mass of blood. On section of that mass of blood you will find it is due

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to disintegration of the tumour. The vessels have given way, and there has been bleeding, and a large accumulation of blood has taken place underneath the skin and fascia. It was because of that bleeding that the patient suffered so much pain, and the pain was caused by the great tension and stretching of the parts.

Many of these tumours do thus break down, and many of them have such hæmorrhages. If you look at these pictures, which are paintings of tumours done soon after their removal, you will see that there are in several of them large blood extravasations, and these swellings may fluctuate and be mistaken for abscesses. But there is something else I want to point out in connection with this specimen. On cutting the bone, though no particular force was being used, it broke across. Here is a longitudinal section taken through the femur, and above the epiphysis there is a fracture. You will see that the bone at the site of the fracture is different from normal bone, and different from the bone of the epiphysis. The latter has not been affected by the growth, and the cartilage of the epiphysis lies as a sort of line of demarcation. The cartilage of the joint also is quite natural and intact. But the bone, where the growth is situated, is completely infiltrated by tumour tissue. There is hardly any osseous material left there, and it is because of the destruction of the bone by this infiltrating tumour that the bone has been weakened and was so easily broken. Now what took place in this case after removal is that which might easily happen during life, and "spontaneous fracture" of a bone is one of the sequences of this form of disease. Here is a picture showing the upper part of a humerus, and you will see some dirty-white growth mottled with some blood extravasations, and at the junction of the upper part of the shaft with the neck of the humerus is a fracture. The bone has been completely destroyed, eaten through by the growth, and it has given way. And you must remember that in a certain number of cases, and in certain parts of the body, a tumour growth may be very little noticed. It may be, indeed, impossible to definitely feel it, and under these conditions it may be that before growth or tumour is ever diagnosed there is a "spontaneous fracture." A good many years ago a patient was admitted into one of our wards on account of constant pain in the gluteal region, in the adductor region and down the back of the thigh. It was thought at first by those who had seen the case to be sciatica. But you should always be suspicious of that diagnosis, because in a great many cases sciatica is a term used as a name for a pain without regarding at all accurately where the pain is situated or what may be the cause of it, and because in a great many cases to which the term "sciatica" is applied it turns out that, though there is perhaps pain in the sciatic nerve, the cause for it is a tumour or abscess. In this woman's case, then, it was thought that the pain was due to sciatica, but examination of the sciatic region did not show any particular tenderness in the line of the sciatic nerve, nor was the pain limited to the sciatic nerve. And the question arose, Had she anything the matter with her hip-joint? It would not move easily, but still it could be moved, and under an anæsthetic it moved very freely indeed. It was before the days of X rays. Nothing abnormal could be felt, and under the circumstances it was thought a wise thing to encourage the patient to use the limb and have it treated by galvanism, and this was done. One morning, however, as the patient was walking about the ward she suddenly fell, with great pain in her hip. She had a spontaneous fracture of the upper part of her femur, and, as it turned out, she had a periosteal growth in an unusual situation, close to the neck of the bone, and consequently buried amongst the big muscles of that region so that it was never felt. The first real evidence of its presence was the occurrence of pain, and the proof that it was sarcoma was the spontaneous fracture. Spontaneous fracture, therefore, ought always to suggest to you the possibility of a tumour growth, and although sarcoma is the common malignant tumour of bone, you must remember that in a certain proportion of cases of malignant disease the disease of bone is secondary to some remote disease of a different nature and of a different part. And it is well recognised that carcinoma of the breast is liable to reproduce itself, especially in the humerus, and the femur, in the sternum, and in the spine. And in either the humerus or in the femur it is by no means an uncommon thing for a spontaneous fracture to be the first real suggestion of there being serious disease of bone. For one of the noticeable things about carcinoma, as compared with sarcoma of bone—and carcinoma you must remember is

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always secondary to disease elsewhere—is that carcinoma will grow into and destroy a bone without forming any external tumour at all, whereas ordinarily a sarcoma forms a very definite tumour, obvious to the patient and usually quite obvious to the sight and touch of the surgeon. On the other hand, in a certain number of cases, carcinoma, even though it is suspected and looked for, will reveal nothing definite to the feel, even when the part is carefully examined. The patient himself or herself in many cases is unconscious of there being anything the matter with the bone until it spontaneously breaks, but in most cases the patient has pain and a sense of weakness, though there may be nothing more definite than that before the tumour finally causes a fracture of the bone, very similar to the fracture which you see in this case. I will mention only two cases to illustrate this. One was that of a patient who believed herself to be quite well and was admitted into the hospital, having slipped in the street close to the General Post Office. When admitted she was found to have a fractured thigh, and no suspicions were aroused at first as to the nature of this fracture. Her history was that of an ordinary street accident. She was put to bed, extension applied, and a bandage put on, and it was not until subsequently, when the Sister of the ward was washing her, that it was found she had something the matter with her breast. She had, in fact, carcinoma of her breast of a slowly-growing type, so-called atrophic cancer, which had not aroused much suspicion in her own mind. In the light of that fact her fractured femur took on a quite different aspect; it was really a "spontaneous fracture" of her thigh-bone as a result of a secondary growth of carcinoma in it.

The other case which I would mention illustrates the difficulty of arriving at a diagnosis. It was that of a patient whose breast I amputated for carcinoma. She was a very thin woman and therefore easy to examine, and about eighteen months after I had removed her breast she complained of what was called "sciatica," that is to say, pain in her thigh. When I came to examine her thigh I could feel nothing, but I told the doctor under whose charge she was that this pain in her thigh and this feeling of weakness and inability to walk made me very suspicious that she might have a growth in her femur, and although I could feel absolutely nothing, a few months afterwards there was a spontaneous fracture of her femur, and she died of dissemination of the disease.

I have said very little about the treatment of these cases of periosteal sarcoma, for it practically always is amputation. You see, if you look at the specimen, that the disease cannot be removed by any other operation. Although it is called periosteal sarcoma that only implies, or ought to imply, that it grows evidently in connection with the periosteum, and the name is given to it in order to differentiate it from the other form of sarcoma of bone which grows inside the bone and is, therefore, called "endosteal"; but you must not imagine that the name "periosteal" means that it is limited to the periosteum. In this specimen you will see that the bone is infiltrated with growth and the muscle also, and it has grown through the tissues up to the skin. However early you might see such a case as that you would never be able to remove the disease by any local operation. The earliest tumour I have ever had the opportunity of seeing was in a boy, æt. 19 years, a well-built, athletic, active fellow, who came into the hospital because he had been kicked at football. He had a very tender place upon his femoral condyle, and I told my house-surgeon that he had probably some extravasation of blood under the periosteum as a result of the kick, and that if he incised it I thought it would probably relieve his pain. This was done next day, and when I came down the house-surgeon told me it was true there was some blood, but when he cut into it there was no definite cavity, and he thought there was a growth which was bleeding, as underneath it he could feel rough bone. If that were so it was evident that the accident was only a superimposed incident—the actual trouble was there before, though the accident had made it worse. Probably the blow upon the sarcoma had caused it to bleed, and in that case also the bleeding caused increased tension and so pain. I explained to the boy that it was a serious thing, and I told his parents that in all probability it meant his losing his leg. They could hardly believe that because he was in good health, active and vigorous, well made and muscular. I operated upon him and found it was periosteal sarcoma. Though so small it had infiltrated the bone already, and it was hopeless to

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remove it locally, so I had to remove it by amputation. I amputated his thigh, but within eighteen months the boy was dead of dissemination of the growth in his lungs, although the tumour of the femur was no bigger than a large walnut.

You may ask, where shall you amputate? In this case I amputated in the upper part of the thigh. My feeling about the site of amputation is that it is seldom worth while to amputate at the hip-joint, for this reason: that apparently there is no greater immunity afforded by amputating high up than there is by cutting through the femur. It is true you must not amputate close to the part of bone affected. If you did so you would find that the growth had extended to that part of the bone and that it would shortly recur in in the stump. These growths generally begin near the knee-joint, so there is a considerable length of bone which you can get at which is healthy, and if you go a good distance away from that joint the results are neither better nor worse than from amputation at the hip. In one respect the results in cases of amputation at the hip are worse, for many more patients die of the operation itself, for the mortality rate from this procedure is far higher than from the other. For that reason, then, I do not think it is worth while to amputate at the hip where an amputation through the thigh will clear away all the local disease and can be done at a place which will prevent any local recurrence. Then there are a certain number of cases in which it is not worth while to amputate at all or, indeed, to do any operation. In the year before last a girl, æt. 17 years, was admitted to Stanley Ward, a tall, well-grown girl with a swelling at the upper part of the femur. It went as high as the small trochanter; it was evidently a periosteal sarcoma of her femur. It could probably be removed, as far as could be ascertained, by amputation at her hip-joint. But I did not do it, I did not operate upon her at all, and I advised that no operation should be done, and for this reason: She had rapidly emaciated, she was very anæmic, and examination of her blood showed a great destruction of red blood-corpuscles, her pulse was rapid and she was in a feeble condition generally. I felt so certain that she had only a short time to live that I decided it was not worth while to amputate at the hip-joint; there was not the prospect of sufficient gain to justify such a procedure in her case. And a noticeable thing about the tumour was that it had caused her no particular pain, so there was not that reason for doing anything. Within six weeks of that time she was dead, probably from dissemination of the growth in the lungs. So you see there are a certain number of cases in which it is not worth while to amputate at all. The question of prognosis after amputation has been dealt with very exhaustively by Mr. Butlin, and I will only quote to you in conclusion what he has to say, after considering the statistics of a good many surgeons: "In sixty-eight cases of periosteal sarcoma of the lower end of the femur there is only one case in which the patient seems really to have been cured by the operation, and when I analysed the account of this case some years ago I could not but doubt whether it was not rather one of central than sub-periosteal sarcoma." So you see out of sixty-eight patients, as far as they could be traced, there was only one, and that a doubtful case, in which the patient had permanently recovered and appeared to be well after an amputation for periosteal sarcoma of the lower end of the femur. And that, of course, indicates that the treatment at the present time for this disease is absolutely unsatisfactory, that the only thing you can do is to operate to relieve the patient's distressing symptoms, and you must remember that there are a certain number of cases in which the patient is so ill and so shortly going to die that it is not worth while to do anything at all.

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PANCREATIC DISEASE.*

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GENTLEMEN,—When your secretary asked me to choose a subject for a lecture, I suggested among others that of pancreatic disease, on the ground that affections of this organ were perhaps less familiar than those of many other parts. The text-books of medicine do not devote much space to this gland, as a rule. Indeed, the study of pancreatic disease is almost a recent development,

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having made more progress in the last ten or fifteen years than during the whole previous history of medicine. One of the pioneers in this field was Fitz in America, while in this country Mr. Mayo Robson has done much to throw light into some of the dark places of our knowledge of this group of affections.

It will be impossible to cover the whole ground of pancreatic disease in a single lecture, so I shall endeavour to deal mainly with the inflammatory affections and some allied conditions, only alluding incidentally to such conditions as tumours and cysts. And you will probably prefer that I should lay stress rather on the clinical than on the pathological side, though it is impossible to separate the one from the other in any branch of medicine.

Clinically, then, we may divide pancreatic troubles into two great groups—the acute and the chronic. To the former belong cases of hæmorrhage into the gland and acute attacks of inflammation; to the latter chronic inflammatory and degenerative conditions, both of which give rise to fibrosis of the pancreas. A good deal of confusion has existed as to the relations of the members of the first group, as acute inflammation in the pancreas is often accompanied by hæmorrhage and has consequently been called acute hæmorrhagic pancreatitis; and on this account some writers are inclined to class all instances of bleeding into the gland or into its neighbourhood under this heading, implying that they are all of an inflammatory nature. But it is just as likely that bleeding may occur without previous inflammation in the pancreas as in other parts, and injury and vascular disease are recognised causes of hæmorrhage elsewhere. It is unreasonable, then, to call all such cases pancreatitis. As a matter of fact, it seems that the pancreas is specially liable to be the seat of hæmorrhage, for if it be examined microscopically after death, we find in a large proportion of all cases that there are small extravasations of blood into its substance without any signs of inflammation. And if small escapes of blood are so common, larger hæmorrhages may well occur in rare instances, due to the same causes, whatever they may be. We have then to deal in the first group with cases in which, on the one hand, there is a sudden hæmorrhage into or around the pancreas, and on the other, with cases of acute inflammation of the gland.

The onset and early symptoms are very similar in both conditions. A healthy man, generally of stout build, who may perhaps have suffered in the past from digestive disturbances, such as abdominal pain and vomiting, or attacks of colicky nature, perhaps accompanied by jaundice, is suddenly seized with excruciating pain in the stomach. He falls into a state of collapse, with a frequent pulse, cold extrêmities, and dusky face. Then vomiting occurs and may persist; first the contents of the stomach are brought up, then bile or bilious fluid, but the vomiting does not become fæcal. The bowels are obstinately constipated. The pain persists, being severe all the time, with exacerbations of greater intensity.

If the abdomen is examined it is found to be distended and tender. The tenderness is greatest in the epigastric region, and some resistance may be found here. The walls move in respiration to some extent, and no visible peristalsis is manifest. In some instances dulness is elicited on percussion of the flanks. The urine is likely to contain a trace of albumen; it may be scanty and show an alteration in the amount of indican present; in rare instances it may contain sugar, and still more rarely acetone and diacetic acid.

The general condition of the patient becomes more and more grave. Delirium may supervene, or marked restlessness be observable. A definite localisation of the abdominal swelling to the epigastric region may be made out. The temperature may be somewhat raised, and excess of leucocytes may be discovered on examining the blood.

A clinical picture, such as I have endeavoured to sketch, is, of course, compatible with more than one affection; all that can certainly be recognised is that some grave accident has occurred in the abdomen, probably in the epigastric region. The affections which will at once occur to the mind of the physician are intestinal obstruction, perforation of the stomach or duodenum, with resulting peritonitis in the upper part of the belly, suppuration in the gall-bladder, and pancreatic disease. It may be useful, then, to consider a little more in detail the features of a pancreatic attack.

In the first place the type of patient is of some interest. Men appear to be more liable to suffer from such conditions than women, though the reason for this selection of the male sex is as obscure as in the many other ailments from which they specially suffer. Taking the whole group of acute

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affections of the pancreas I find that in the last eleven years 116 cases are recorded in males against 66 in females, or not very far from 2 to 1. Then a second noticeable feature is the tendency of fat persons to fall victims to pancreatic disease. Perhaps a quarter of all the cases arise in stout patients. The reason for this, again, is quite obscure. Many fat persons have, it is true, degenerate arteries, so that a tendency to bleeding might be thus accounted for, while fatty degeneration of the pancreas itself, which is common in such persons, might lead to rupture of vessels owing to diminution in the support afforded them by the surrounding tissue of the gland. But the tendency of stout persons to suffer from inflammatory affections of the pancreas would not be thus explained. Now, for some reason fat persons are specially subject to formation of concretions in the bile-passages—to gall-stones, in short—and some recent observers, notably Opie in America, have attached much importance to gall-stones as a cause of pancreatic disease. It is suggested that a gall-stone may become impacted in the orifice of the papilla through which both the biliary and the pancreatic ducts enter into the duodenum, and that when this occurs, bile, being prevented from entering the gut, is forced backwards along the ducts of the pancreas and by its irritant action sets up inflammatory changes in the gland, which may even be hæmorrhagic in intensity. Of the possibility of the bile acting in this way there seems no doubt, as experiments on animals prove that it has this irritating quality; but the number of cases of pancreatic disease in which gall-stones are found is scarcely so large as we should expect if this were the usual mode of causation, and the proportion of those in which an impacted stone is found in the papilla is infinitesimal. It is quite equally possible that the same cause which gives rise to the gall-stone may also cause the pancreatitis, as we now believe that infective bacteria are the main excitants of the catarrh of the bile-ducts which gives rise to gall-stones. Such an explanation would agree better with the actual findings at necropsies, in which, as I have said, the presence of gall-stones is the exception rather than the rule. We must, however, remember that these concretions may easily be overlooked, and may be present in reality more frequently than they are recorded.

We have thus in the type of patient generally affected some assistance in excluding some of the conditions which may be mistaken for pancreatitis. A gastric or duodenal ulcer, for example, generally leads to considerable and prolonged digestive disturbance, and hence to emaciation. A fat person then is not, generally speaking, likely to be the subject of this affection, and therefore is not likely to suffer from perforative peritonitis. A little while ago one would have further said that gastric ulceration was much commoner in women than in men, and would have used this also as some criterion as to the probability of perforation as against pancreatitis. But the recent view appears to be that the two sexes suffer almost equally from actual ulceration of the stomach, the great number of young women who suffer from dyspepsia, and even from hæmatemesis, having no organic lesion. This view is founded on post-mortem findings, which are at least reliable so far as they go. At the same time one may be excused for exhibiting some degree of nervousness as to the propriety of administering solid food after the occurrence of hæmatemesis, in case an ulcer should be present. This, however, is a digression.

After the study of the character of the patient, the next point that claims attention in acute pancreatic disease is the character of the pain. This is usually excruciating in intensity in cases in which there is much hæmorrhage. The patient rolls about in agony or seems doubled up by it. It is generally epigastric, but may be referred to the præcordium, or to the right iliac fossa. Sometimes it is very severe in the loins and back. In one recorded case pain was complained of running down the thighs, like sciatica. The abdominal pain is apparently much more severe than that of perforative peritonitis, and it seems often to resist the effects of morphia given for its relief. Along with the pain there often goes a condition of restlessness, the patient turning from one position to another, sometimes actually getting up and moving about the room—a very different state from that of peritonitis, in which the sufferer generally lies as quiet as possible, with drawn-up knees, endeavouring to keep the abdominal walls still. Marked delirium may be a feature of pancreatic cases, and this is rather a distinguishing characteristic, as contrasted with most of the conditions with which they may be confused.

The collapse met with in acute pancreatic

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conditions has nothing characteristic, but there is not infrequently marked cyanosis of the face and extremities. The vomiting, again, presents ordinary features, not being fæcal in character. This last is a point of importance in the diagnosis from intestinal obstruction, in which this sign occurs as a rule before long, unless the obstruction is very high up in the small intestine. In pancreatic disease the vomiting often lessens as time goes on, whereas in obstruction of the bowel it usually becomes progressively worse. Hæmatemesis may occur, the blood presumably reaching the duodenum through the pancreatic ducts.

The resemblance of the cases which we are considering to intestinal obstruction is increased by the obstinate constipation by which they are accompanied. The reason for this is not very clear, but there is probably a reflex spasm of the colon, this condition having been actually found post-mortem in one case of acute pancreatitis. It is possible that in some instances the swollen head of the gland may press upon the duodenum; there may also be some peritonitis over an inflamed pancreas, and this may spread to the intestines with resulting paralysis of the muscular coat. The constipation may disappear as the case progresses; it may even be replaced by diarrhœa in some instances. Rarely the motions may be altered in character as we shall see that they are in chronic cases.

The urine is generally scanty, and may be entirely suppressed at first. Glycosuria is uncommon, but when it occurs it is a valuable indication of the condition present. It was met with ten times out of 92 cases of hæmorrhage into the gland, and the same number of times among 102 cases of acute inflammation and necrosis of the pancreas. The following is a case which I have recorded elsewhere:

A laundress, æt. 53 years, was admitted into Charing Cross Hospital under the care of Dr. J. M. Bruce on January 24th, 1893, complaining of abdominal pain, thirst, and dyspnœa. Her previous history had been uneventful; there was no history of syphilis. A week before admission to hospital she was seized with pain in the abdomen, which rapidly swelled and became hard to the touch. At the same time she complained of increased thirst and of shortness of breath, and had a difficulty in getting about. On examination she appeared to be a well-nourished woman; her face was flushed, and her tongue was dry and covered with brown fur. Her temperature was 100° F., and she complained of great thirst. There was pain all over the abdomen, which was uniformly distended, so that the umbilicus was obliterated. Tenderness was general over the abdomen, but there was increased resistance in the epigastrium and hypochondria, especially on the right side. The bladder was distended, and the edge of the liver could be felt about one and a half inches below the costal margin. She was delirious at night, trying to get out of bed continually. She passed from 54 to 60 ounces of urine *per diem*, the total amount of sugar present varying from 1012 to 1125 grains. Her temperature gradually rose, and on the 29th and 30th she had rigors. On the latter day acetone was noted to be present in the urine. She vomited and became very restless. Finally she died in collapse without any appearance of coma. At the necropsy there was found "a good deal of turbid fluid in the peritoneum; some little lymph between the coils of the intestines." The layers of the mesentery were everywhere separated by a large mass of disintegrated blood-clot and blood-stained fluid. The stomach was adherent to the left lobe of the liver, and on separating the two several pints of blood-stained grumous fluid escaped. "The fat was everywhere speckled over with opaque, dead-white points, which appeared to be composed of necrosing fat. . . . In the situation of the pancreas was a breaking-down mass of tissue along with much bloody fluid. Some loose shreds of tissue, as big as a fist, were floating in this cavity; they appeared to be portions of the pancreas." No other morbid conditions were found.

In this case there had been no symptoms of diabetes before the onset of acute symptoms. The pancreatic condition was therefore responsible for the glycosuria and acetonuria.

Pancreatic hæmorrhage may occur after operations on the biliary passages and cause a fatal termination, as in the case of a man, æt. 54 years, who was operated upon in Charing Cross Hospital for stricture of the bile-duct, supposed to be due to an impacted gall-stone. He died almost suddenly a day or two after the operation, and at the necropsy, which I made, there was a large effusion of blood around the pancreas, the substance of which was intensely fatty and so torn up by the

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blood that it was difficult to find portions of it for microscopical examination. The obstruction of the bile-duct proved to be due to a ring of malignant disease arising in the duct itself.

Clinically, it is practically impossible to distinguish at first between cases due to hæmorrhage into the pancreas and those due to acute inflammation. In the former, however, the onset is usually more abrupt, and the pain more agonising at first. Collapse also is more marked at the very beginning. The occurrence of hæmatemesis or melæna, should either occur, will indicate this accident. Death may ensue in a few hours in this affection; the average period is probably about four or five days. Deceptive periods of apparent improvement sometimes occur. In the most acute cases of inflammation, in which actual gangrene of the pancreas occurs, the duration may be almost as short, but in cases of average severity life is likely to be prolonged for two or three weeks, and in suppurative cases even for months. Time is thus afforded for the appearance of a definite abdominal tumour, which generally occupies the epigastric region, lying rather to one or other side of the mid-line, when pus has formed, according as the head or tail of the gland is the chief seat of disease.

Hæmorrhage into the pancreas is one cause of the formation of pancreatic cysts. In cases of old hæmorrhage I have found the gland studded throughout with minute cysts scattered through the connective-tissue stroma. It is possible to see the formation of rounded cavities in the blood-clot, the fibrous wall of the cyst being produced by the inflammatory reaction of the surrounding tissue. Truhart believes that practically all pancreatic cysts are formed in this way, but it is generally held that some are retention cysts, due to accumulation of secretion behind an obstruction, while others are cystic adenomata, like ovarian cysts. Hæmorrhage may occur into a cyst after it has formed.

It seems now demonstrated beyond the possibility of doubt that an acute inflammation of the pancreas may occur in the course of an attack of mumps, analogous to the epididymitis which is well known to ensue. The condition is marked by vomiting and epigastric pain arising in the course of the parotid affection. A more or less definite tumour may be made out on palpation in the epigastrium, which does not move on respiration. The attack lasts about a week, subsiding gradually. So far all the cases have recovered satisfactorily, but the condition is rather alarming, and the possibility of the onset of pancreatitis must be borne in mind in treating cases of mumps.

If a patient has died of one of the acute conditions included in this first group of affections, the conditions found at the autopsy will vary in different cases. If hæmorrhage has occurred there will be an extravasation of blood into the substance of the pancreas, which looks dark red and much enlarged, while the root of the mesentery and the retro-peritoneal areolar tissue will also be infiltrated. Blood may be found in the intestine, passing probably along the pancreatic ducts. The peculiar feature known as fat-necrosis is usually present; it will be convenient to consider it separately a little later. Extensive necrosis of the pancreas itself often results from hæmorrhage, the nutrition of the gland being interfered with by the pressure of the extravasation within its capsule, and its tissues at the same time being torn up by the escaping blood.

In inflammatory conditions the pancreas looks swollen, and its capsule is tense; it has been compared to a pudding tied up in a bag, or even to a plaster-of-Paris cast, owing to its increased hardness. It may be seen on section to be studded with hæmorrhagic areas, or may look almost normal in colour, as the hyperæmia which existed during life is not visible after death. Softened areas of necrotic tissue may be visible. On microscopical examination the tissue will be found infiltrated with leucocytes, much of the glandular substance being disorganised and failing to take up the stain employed. If suppuration have occurred, there may be a single abscess occupying one part of the pancreas, or the whole gland may be riddled with suppurating tracks, so as to resemble a sponge full of pus. The pus may escape into the lesser sac of the peritoneum and form a large collection here, or it may track behind the peritoneum in front of one or other kidney. It may even perforate the intestine from without, and in one case it escaped *per vaginam* during life.

When collections of fluid occur in the lesser sac of the peritoneum or in the neighbourhood of the pancreas, it may be possible to demonstrate in it the presence of pancreatic ferments, trypsin, amylopsin, and steapsin or lipase. One or more of

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these bodies may be wanting, while the others are demonstrable.

The cause of death in acute disease of the pancreas is not quite clear. In the case of hæmorrhage shock and actual loss of blood may contribute to the fatal issue, but neither seems to be the actual cause of death, for the amount of blood effused is not often so great as to be in itself fatal, and death is delayed till the first shock has passed off. It would seem that some toxic body is formed and absorbed into the system. It has been held that this is trypsin itself, but this is doubtful. As is well known, trypsin can be injected in small doses, as has been done for the treatment of malignant disease, without producing any bad results. A very large amount must be assumed then to be absorbed in pancreatic affections if it is to cause death. But it must also be remembered that the pancreas itself contains only protrypsin, the forerunner of trypsin, an inert body until it meets with some second substance capable of rendering it active. It is not clear whence the latter is derived in these cases, though the ingenious suggestion has been made that the cause of pancreatic hæmorrhage is the entrance into the pancreatic ducts of intestinal juice containing the activating substance, enterokinase, whereby active trypsin is formed within the gland and causes serious injury.

The question of treatment resolves itself into that of operation or no operation. In this respect the cases accompanied by suppuration stand apart from the others, inasmuch as no one can doubt that here operation is necessary for the evacuation of the pus, as would be done in other parts of the body. And it is in suppurative cases that the results of operation are the most favourable. Among forty-five recent cases of suppurative pancreatitis there were twenty-three deaths; thirty-five were operated upon, and of these thirteen died, or about 37 per cent.; of those not operated on (ten) none recovered.

In cases of hæmorrhage into the pancreas and its neighbourhood, it may be argued that operation is not likely to do much good. It is seldom, if ever, possible to find a bleeding point and tie the vessel so as to arrest the hæmorrhage, while it seems doubtful whether the actual loss of blood is accountable in any considerable proportion of cases for the fatal issue. It might seem then that the patient's chance of recovery would be diminished rather than increased by opening the abdomen. A study of actual cases, however, seems to prove that laparotomy is the proper treatment, as a fair number (sixteen) cases of recovery are now on record in patients who were operated upon, whereas the mortality among those not operated on is very great. It is impossible to say exactly what it is as without operation an exact diagnosis is only possible after death. To say, then, that all the cases not operated upon died, though true as far as the records go, would be misleading, as recoveries could not well be recorded. It is certain that mild cases of hæmorrhage into the pancreas do recover, as traces of the condition are found, as I have already pointed out, post mortem, in cases in which the condition had not been suspected during life. If, however, we group together the cases in which acute inflammation of the pancreas existed or was diagnosed, and those of hæmorrhage, we find that among a total of 194 cases 54 recovered (28 per cent.), while of 54 cases of hæmorrhage operated upon 16 recovered (29·6 per cent.). The mortality is therefore rather less in these very acute cases after operation than in the whole number of acute pancreatic disease recently reported (so far as I have been able to collect them), both mild and severe, operated and not operated upon. Among the latter are included some cases of comparatively slight severity, such as those accompanying mumps. The conclusion would seem then to be, that in cases of severe symptoms referable to the pancreas, we should open the abdomen. The advantage gained in case of hæmorrhage would seem to be the removal of the mixture of blood and pancreatic secretion, which may be in itself toxic. It is also possible to incise the pancreas itself, if its capsule is so tightly distended with blood as to threaten necrosis of the gland, and so avert this danger. This latter possibility is probably the strongest reason for operating in cases of acute inflammation before pus has formed, for there is no doubt that necrosis of the gland is the most fatal of all pancreatic lesions. An additional cause for operating lies in the fact that exact diagnosis is rarely possible on clinical symptoms alone, and the conditions with which pancreatic disease may be confounded, such as perforative peritonitis, gall-stone colic, and intestinal obstruction, are all such as to demand operative interference.

As to the method to be adopted in operating I

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am of course not competent to speak. Different surgeons have taken different routes to reach the gland, some making an incision in the front of the abdomen, others in the loin, and others again preferring to resect a rib and enter through the diaphragm. In those cases in which there is co-existent empyema or a suspicion of it, the last method has obvious advantages. It is noteworthy that its chief advocate, Guinard, in France, has three cases of recovery to record, and believes that this is the method of election in all instances. Probably the anterior abdominal route will be generally adopted, in view of the difficulties of diagnosis, a counter-opening being made in the loin if it appear desirable.

When we come to the second group of cases, those of chronic disease of the pancreas, we are met by a very different clinical picture. If the acute diseases of the organ offer one of the most alarming abdominal conditions which can be encountered, the chronic cases are distinguished chiefly by their indefinite character and obscure symptoms. It must be confessed that very few of all cases of fibrosis of the pancreas are recognised during life. I have now examined over 250 pancreases taken from cases dying in hospital, and find that a certain amount of fibrosis of the gland—analogous to cirrhosis of the liver—is present in about 15 per cent. of the whole number. It is commoner in persons over forty years of age than in younger persons, and would seem to be due in the former to a gradual degenerative process in which the glandular tissue wastes and is replaced by connective tissue. It does not seem to give rise to any symptoms in most instances, though the greater frequency of diabetes in elderly persons may be related to this fact. To discuss the relation of this disease to atrophy and degeneration of the pancreas would lead us too far afield. In younger persons syphilis and cirrhosis of the liver are probably the two commonest causes of pancreatic fibrosis; or, more accurately, the condition which causes cirrhosis also produces a similar condition in the pancreas. Syphilitic fibrosis is not uncommon in infants who die in the first few days or weeks of life from congenital syphilis. In a child, æt. 7 years, who died with symptoms of severe tetany, and whose pancreas was sent me for examination by Dr. Jollye, of Brixton, there was intense syphilitic fibrosis of the gland; this had been the only organ apparently diseased, so far as could be seen at the autopsy. Whether disease of the pancreas can produce tetanoid convulsions I cannot say, though convulsions have been noted in one or two cases of acute disease of the pancreas. Possibly the cause which produced the tetany, which is generally attributed to the absorption of poisons from the stomach, also produced the fibrosis of the pancreas, inflammation spreading up the ducts of the gland from the duodenum.

The cases of chronic disease of the pancreas which may be recognised during life are of a somewhat different type. The main features are wasting, progressive weakness, jaundice, and attacks of abdominal pain. A characteristic case would be somewhat as follows: a man, of middle age, has suffered from frequent attacks of abdominal pain, with vomiting and anorexia; in the last attack he became jaundiced and the jaundice has persisted since. The pain has meanwhile become easier, but has recurred at intervals; he may have bouts of shivering, almost deserving the name of rigors. He has got much thinner during his illness, and complains of great weakness. His appetite has been so bad that he may have felt a positive loathing for food of all kinds. Jaundice has persisted for some weeks and appears to be deepening rather than diminishing. The motions are frequent and loose, pale in colour and offensive in odour. On physical examination it may be possible to feel a distended gall-bladder, and in addition to this there may be a sense of resistance, or even a palpable mass in the epigastrium, in the region of the pylorus. The usual phenomena accompanying jaundice, slow pulse and itching of the skin, may be present. In such a condition as that just described probably the first idea that will occur to the mind of the physician is the existence of malignant disease in some part of the digestive tract, either in the stomach with secondary involvement of the liver or of the glands in its hilum, or of the pancreas. Enlargement of the liver, which occurs in some instances, makes such a diagnosis even more seductive. Jaundice due to impacted gall-stone may also be accompanied by some wasting, but not to the same extent as in a typical case of pancreatitis. Certain special phenomena may be met with in chronic disease of the pancreas, which, taken together, may enable a diagnosis to be made of some affection of this organ. The most striking

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and constant are alterations in the character of the stools, while there may also be pigmentation of the skin, and the presence of sugar in the urine.

The pancreatic juice, as is well known, contains ferments which act upon the three main groups of food-stuffs—proteins, fats, and carbohydrates. It might be expected, then, that if the gland were diseased metabolism of all these would suffer. So, indeed, it is, but they are not all affected in the same way. The carbohydrate metabolism is mainly affected in a manner which is not understood, and the result is the appearance of the condition known as diabetes in a certain proportion of cases. This seems to be due to lack of some internal secretion of the pancreas, or, at all events, of some influence exerted on the manufacture or disposal of carbohydrate material in the body. It is not a direct effect of absence of pancreatic juice from the intestine. The digestion of starchy matters in the alimentary canal probably proceeds almost as usual in diabetes; it is the assimilation of the sugar produced or some abnormal production of sugar which is at fault. It would lead us too far to discuss this question here. At all events the fæces do not appear seriously altered in respect of their carbohydrate content; perhaps the intestinal secretion is capable of taking the place of the pancreatic amylopsin.

The most obvious failure in chronic pancreatic disease is in the digestion of fats. The stools are bulky, pale, and greasy; the presence of fat in them may be recognisable with the naked eye, and the microscope reveals the existence of abundant oily droplets. A rough estimation of the amount of fat present may be gained by drying and weighing a portion of fæcal matter, and then extracting with ether and weighing the residue. For exact computations laboratory methods are necessary, by which we can determine, not only the amount of fatty material present, but also the relative proportions of neutral fat and fatty acids, the relation between which is altered, more of the former and less of the latter being found in cases of pancreatic disease owing to the absence of the fat-splitting ferment from the intestine. Normally it is said that 75 per cent. of the fat is split up; in pancreatic disease only some 25 per cent. may be thus altered. It is necessary to bear in mind that the absolute amount of fat present in the fæces depends on the amount taken with the food, and that, therefore, for purposes of exact estimation it is requisite to put the patient on a diet containing a known amount of fat.

Defect in digestion of meat is easily recognised on microscopical examination of the stools, muscle-fibres being found which have preserved their original striation. For the recognition of a failure to digest nuclei Schmidt has devised a special procedure. He hardens small cubes of meat in alcohol and ties them up in little silken bags, the strings used for tying being left long, so as to facilitate the finding of the bags afterwards. When they appear in the fæces they are taken and washed, and then stained, cut into sections, and examined microscopically to ascertain the state of the nuclei. It is necessary that the test-substance should not be unduly hurried through the alimentary canal, as by diarrhœa, as in that case even normal intestinal ferments may fail to break up the nuclei. Nor, on the other hand, must their sojourn in the gut be unduly prolonged, or decomposition may occur, due to putrefactive organisms, and the effects of digestion may be simulated.

Of the jaundice which occurs in many of these cases, and of the value of Dr. Cammidge's test, I will say no more here, as you will be hearing of them from Mr. Mayo Robson himself a little later on.

Alterations in the urine may be found, especially the presence of sugar in a certain proportion of the cases. Occasionally maltose is present instead of the ordinary glucose of diabetes. Glycerine has been found in the urine by Testi. Dr. Cammidge who originally examined the urine in these cases for glycerine, has found that pentose—a sugar with five instead of six atoms of carbon—and sometimes glycuronic acid are present in the urine, and give crystals when treated with phenyl-hydrazine, which are characteristic of pancreatic disease. The details of the test are somewhat complicated and need not be described here. Mr. Mayo Robson holds that the test has considerable value for purposes of diagnosis.

Cases of chronic pancreatitis in which all or most of the characteristic symptoms are present—jaundice, wasting, alterations in the stools and perhaps in the urine—are not common. When they do occur, there is not much difficulty in the diagnosis, except that the distinction between simple pancreatitis and cancer of the pancreas is difficult: there can be little doubt as to the organ which is at

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fault. The cases about which doubt arises are those in which only some of the symptoms are found or in which detailed examination is not feasible. One such which I recently saw was the case of an elderly Jew, who came into Charing Cross Hospital for abdominal pain. He did not seem very ill at first; his abdomen was a little swollen, and there was apparently a small amount of free fluid in the peritoneal cavity. His skin was dark and covered with freckles. The fluid in the abdomen, if there really ever was any, disappeared in a few days' time, but the patient did not improve. He grew weaker and thinner, and still complained of some abdominal pain. No tumour or other abnormality was ever palpable in the belly. The stools presented nothing noteworthy to the naked eye. Then one day the clinical clerk found that there was a distinct trace of sugar in the urine, but only on one occasion. Attention was naturally directed to the pancreas by this discovery, and I was anxious to have a microscopical examination made of the fæces, and to have Dr. Cammidge's test applied to the urine. Unfortunately at the time our Clinical Pathologist was away, and while we waited for his return the patient suddenly insisted on going out of hospital. So the diagnosis remained problematical.

A second case which also came under my observation lately was that of an elderly man, a builder by trade, who had been ill for about a year. He had attacks of severe abdominal pain at intervals, and had wasted rapidly. His wife thought that his skin had become darker lately, and stated that on one or two occasions his stool was frothy and offensive. The urine was normal, containing neither sugar nor albumen. Nothing definitely amiss was discoverable in the abdomen, but there was marked tenderness almost everywhere on palpation. The presence of a few râles at the apex of the left lung raised a suspicion of tuberculosis, but there was no cough or other sign of phthisis. Here the wasting, abdominal pain, and (?) pigmentation of the skin were in favour of pancreatic disease; the absence of jaundice and of any constant alteration in the stools, as well as the diffuse tenderness of the abdomen, were against it. I suggested the possibility of pancreatic affection to the doctor who sent me the case, and recommended an examination of the fæces, but I have not heard any further news of the patient.

The question of the surgical treatment of chronic pancreatitis has practically only arisen in the last few years, chiefly owing to the work of Mr. Mayo Robson. This treatment is applicable to cases in which there is marked jaundice, and consists in performing one of the recognised operations for the relief of retention of bile—viz. opening and draining the gall-bladder (cholecystotomy) with or without formation of a permanent fistula leading to the surface (cholecystostomy) or of an artificial connection between the gall-bladder and the gut (cholecyst-enterostomy). You will, no doubt, hear from Mr. Mayo Robson all that is to be said as to the rules for such procedures. But there are undoubtedly a considerable number of cases in which the pancreas is the seat of fibrosis, and yet jaundice is absent. They are not common, or at all events they are not often recognised, but probably as more attention is paid to the subject the condition will be found to be not so very rare. In such instances there appears no reason for surgical interference, but we have to do what we can to relieve the disturbances arising from lack of pancreatic juice. It is only in rare instances that we can hope to influence the condition of the gland itself, namely in the fibrosis which is due to syphilis; here the administration of iodide of potassium may cause the disappearance of symptoms, such as glycosuria, and lead to practical recovery. In other instances something may be done by artificially supplying the pancreatic ferments. Several preparations are obtainable under the names of pancreatin, pankreon, etc. The last-mentioned is supposed to be insoluble in gastric juice, but to dissolve in the pancreatic and intestinal secretions. Hence it is claimed that it will reach the duodenum unaffected by the gastric juice, which may tend to destroy other preparations. Good results and failures are recorded from both these remedies, and more experience is needed before we can properly judge of their utility. At all events some of them are worth trying if we have reason to suspect pancreatic insufficiency.

The diet, too, must be regulated according to the condition present. Fatty foods are generally to be avoided, especially if the stools show the passage of much fat; carbohydrates should also be diminished, while proteins, which are digested to a large extent by the gastric juice, are admissible.

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There does not seem much evidence available as yet as to the value of Professor Starling's secretin as a remedy in such cases. One or two writers have claimed some slight benefit from it in cases of diabetes, but in two cases admitted to Charing Cross Hospital, under the care of Dr. F. W. Mott, F.R.S., in which it was adminis. tered—cases which I had the good fortune to have charge of during his absence—no good results were descernible; in one, indeed, the patient declared himself better when the remedy was discontinued. Perhaps these were not quite fair cases in which to test the value of the remedy on the pancreas, as in both after death there was found advanced atrophy of the organ, so there was little tissue left for the secretion to influence. In one case the stools showed some excess of fat and some undigested muscle-fibres, being also much increased in quantity, so that the patient complained of diarrhœa, meaning thereby frequent call to stool, but no influence on this digestive abnormality could be traced to the secretin. However, cases of diabetes do not properly fall within the scope of our present subject.

In connection with the study of disease of the pancreas I ought to make some allusion to that peculiar condition which is often found *post mortem* in cases of acute lesion of the gland, fat-necrosis. When the abdomen is opened, either at an operation during life or after death at the necropsy, the peritoneum is seen to be dotted with small whitish points, from 1 mm. to perhaps 5 mm. in diameter. On section they are seen to be somewhat of the shape of a split-pea, presenting a rounded contour on their deep aspect. They are soapy in consistency and can be scraped out with the point of a knife. They are most numerous in the neighbourhood of the pancreas, and not infrequently points of fat-necrosis may be found in the fat that infiltrates this organ when there is no sign of the condition elsewhere. In other instances there are points of fat-necrosis at considerable distances from the pancreas; they have been found in the subcutaneous fat, in that beneath the pericardium, and even in the marrow of the bones.

Balser, who first described the condition, finding it present in fat persons (who, as previously mentioned, are the favourite subjects of pancreatic disease), suggested that it was a necrosis arising from the overgrowth of the fat, some parts of which failed in consequence to receive sufficient nourishment. Lancereaux next found that the process at work was a saponification of the fat, by which it is split into glycerine and fatty acid. The association of fat-necrosis with pancreatic disease taken along with the fact that the pancreas forms a fat-splitting ferment, naturally suggested that it was to the action of this substance that we must attribute fat-necrosis.

With regard to the actual affections of the pancreas which are associated with fat-necrosis, it was present in 56 instances among the 92 cases of hæmorrhage which I have collected. Among 102 cases, including necrosis of the gland, suppuration, and simple acute inflammation, it occurred 48 times, while it was only noted twice among 60 cases of chronic pancreatitis. On the other hand, it is only in very rare instances that fat-necrosis is found apart from noticeable lesions of the pancreas. Perhaps a dozen cases altogether have been recorded. In several of these, however, it was noted that there was blood in the peritoneal cavity, and the frequency of bleeding from or into the pancreas makes it, at all events, probable that in these instances there was some minute lesion of the gland which could not be discovered. A small breach of continuity of its surface would suffice to allow some of the juice to escape, but might easily elude observation while the histology of the gland might be quite normal otherwise. Further, the actual fat-splitting ferment, lipase or steapsin, has been found in the fat-necroses, and it is impossible to suggest any other origin for it than the pancreatic juice.

Difficulties have been raised to this pancreatic theory of fat-necrosis on account of the occurrence of necrotic points in distant parts, such as the pericardium and even the bone-marrow. An explanation has recently been given of this apparent difficulty. It has been found that when the pancreas is injured some of the glandular cells may be carried away by the blood-stream and lodged as emboli in other parts. These embolic cells contain enough ferment to produce fat-necrosis where they are impacted.

The chief importance of fat-necrosis is that if it is observed at an operation evidence is at once afforded that the pancreas is in some way affected. Now and again one reads of cases of pancreatic disease which were not recognised even at the operation, the pancreas not being examined. This examination should always be made if any points of fat-necrosis are visible. Indeed, it is well to bear in mind the existence of this gland in all cases of urgent abdominal disease, so that it may be scrutinised if there is not adequate evidence of disease elsewhere.

February 3rd, 1908.

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COLI-BACILLURIA.*

By H. BATTY SHAW, M.D., F.R.C.P.,

Physician to University College Hospital; Lecturer in Therapeutics University College Hospital Medical School; and Assistant Physician to the Hospital for Consumption and Diseases of the Chest, Brompton.

THE study of bacterial invasion of the urinary tract has until comparatively recent years attracted the attention mainly of the surgeons, and little notice has been given to the subject by physicians until the occurrence of typho-bacilluria attracted inquiry. Besides being a tract which is invaded by organisms largely owing to the instrumental investigation and treatment of the lower urinary passages, including the urethra and bladder, it is now more clearly obvious that the urinary tract may be one by which organisms are extruded from the system after they have gained access to it by some other route, *e. g.* it is well known that the staphylococcus, streptococcus and other organisms which have brought about a general invasion of the system may escape by the urinary tract, be found entrapped by the kidney and be lodged in its interstices. Even the diphtheria bacillus, which usually merely grows on various parts of the upper respiratory tract and does not invade the system, may find access to the kidney substance. It remains for further clinical and pathological investigations to differentiate those cases in which these orgunisms are being merely extruded from those in which it is probable that during such extrusion they exert a pathogenic effort. Already it is very well known that the tubercle bacillus, possibly during the process of extrusion, may become a most harmful denizen of the kidney, ureters and bladder, and judging from the scanty recognition at the present day of the harm that can be done when the coli-bacillus

* Being a clinical lecture delivered at University College Hospital.

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(*Bacillus coli communis*) gains access to the whole or part of the urinary tract, it is well for the physician to study the possible consequences of such an invasion in cases which, from various reasons, are relegated to him alone or in partnership with the surgeon. Already it is well recognised by both medical and surgical writers that the coli-bacillus is able to cause suppuration in and around the kidney, and that it may cause such severe suppurative changes in the bladder as to be looked upon as a potent cause of suppurative cystitis, but medical text-books are silent, or almost so, upon the important subject of coli-infection of the urinary tract which is *not* accompanied at least by coarse naked-eye evidence of suppuration. Whether the infection is located in the kidney or kidneys only, or in the lower part of the urinary tract only, provided that gross suppuration is not a feature of such infection, there can be little doubt that too often this infection, as well as the infection by other pathogenic organisms, are overlooked or treated as trivial.

As proof of the latter statement the following brief records of cases are given, and it will be understood that these cases were for some time not properly diagnosed during life, and even in some cases were not recognised till the post-mortem investigation revealed how infection, and especially coli-infection, had masqueraded during the life of the individual in a protean mimicry of other better known forms of disease.

Clinical Examples of Urinary Infection.

(1) An elderly man who had been a labourer in the dockyard at Chatham came under observation because he had suffered from elevation of temperature which was intermittent in type. No cause could be found for the temperature, but it was suspected that he had caught malaria owing to his work on board ships which had voyaged to parts of the world where malaria was known to exist; blood examination was, however, negative. Subsequently a special examination was made, and after suitable treatment the patient left the hospital quite free from temperature.

So closely similar was the febrile reaction of the patient to that met with in malaria that the clinician in charge of the case was confident of his diagnosis, and arranged for the special examination for the hæmamœba of malaria. The urine under ordinary routine examination had revealed no feature which suggested any involvement of the urinary tract, and it was only after the patient had been discovered to be surreptitiously passing a catheter to overcome the difficulties of urination, consequent upon the presence of an enlarged prostate, that the true state of affairs was recognised. His urine was loaded with motile bacillary elements, probably introduced into the urinary tract as a result of instrumentation. It is quite true that this is but an example of a common surgical malady, but the fact remains that the surgical nature of the disorder was overlooked and the patient was handed over to the physician for diagnosis and treatment.

(2) A man, æt. about 40 years, who had knocked about the world a great deal, had been ordered home to England in order to shake off recurrent attacks of fever. These attacks of fever were intermittent in character, and he was sent into the hospital in order to have his blood examined for the malarial parasite. As in Case 1, with suitable treatment the temperature fell and he was discharged relieved.

The experience of the first case led to an inquiry for the possible disease of the urinary tract, and from the patient's locker was abstracted a well-worn bougie with which the patient at regular intervals of a month had dilated a stricture of some years' standing. The urine was found to be free from the abnormalities usually detected by ordinary clinical investigation, but as in Case 1 bacteriological investigation showed that he had infected himself with a motile organism, though coarse signs of suppuration were not in evidence. The usual surgical treatment prescribed for such cases resulted in the disappearance of the fever and its accompanying symptoms.

(3) An elderly patient suffered from hæmaturia. Exploration by sounding was carried out. He had a rigor a few hours afterwards and acute cystitis set in. The temperature was elevated and remained so for some weeks. During this period he became semi-comatose and passed blood and slime in the stools, which were very frequently evacuated and caused pain. The tongue became dry, thrush developed in the mouth and the patient became very seriously ill. He, however, rallied, and eventually became restored to complete health; the urine, which had during the cystitis shown a good deal of albumen, became free from proteid. When allowed to stand it became

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very offensive, but remained acid. There was no evidence that the escape of urine during micturition was impeded by the presence of a stricture or of a tumour, or of an enlargement of the prostate. Despite the fact that this patient was able to return to his ordinary duties, he has never lost the coli-bacillus from his urine, nor has he for quite a considerable time—about four years—ever suffered from elevation of temperature, except on two occasions, in which tenderness developed in the left epididymis. After the preliminary catheterisation, and on the onset of the diarrhœa and other manifestations of disturbances of the bowel, it was conjectured that instrumentation had led to the development of a fistula between the bladder and rectum, and that from an interchange of the normal contents of bladder and bowel, cystitis and mucous colitis had been set up. As the subsequent history showed, coli-infection of the bladder had taken place, but the colitis was probably of the same nature as the enteritis, etc., which is known to occur as a remote consequence of bacterial infection of some other organ than the original one involved, comparable to the enteritis so often met with in acute pneumonia. Despite every effort, both local and general, no treatment has succeeded in removing from the urinary tract the organisms which had fastened upon the bladder wall.

(4) A girl, æt. 14 years, under treatment for bronchiectasis for several years, has during the last two months been afflicted with persistent frequency of micturition and enuresis, and the only other feature noticed by the mother was that the urine was highly offensive; it contained a minute trace of albumen. The frequency was usually unaccompanied by pain, though occasionally this symptom was present in a slight degree. The urine was found to be loaded with the coli-bacillus. An ordinary naked-eye examination showed that after standing, a deposit of what quite easily might be taken for a slight excess of mucus (physiological in origin) had taken place, and the upper part of the urine became quite clear. Without any effort on the part of the physician the coli-bacilluria diminished, though it did not disappear, and the symptoms entirely disappeared. At no time during observation was the temperature found to be raised, nor were there any signs of general symptoms, such as loss of appetite, etc. The case had originally been diagnosed as one of bronchiectasis (of which there were obvious signs) and of enuresis of childhood, probably due to weakness of the spinal centres for micturition, etc. On further inquiry it was found that the child had been subject to previous attacks of enuresis, which, however, subsided spontaneously; no local abnormality of the genitalia could be detected.

(5) A man, æt. about 50 years, had been under observation for bronchitis and emphysema and occasional attacks of asthma. Periodically he had had attacks of "fever and gout." During these attacks he had shivered and was troubled with fleeting pains about his body, limbs and small joints; he suffered from stricture of the urethra, and was negligent of treatment; the urine (naked eye) free from purulent deposit, was acid, and on occasions was offensive. He was born in India, has resided in England twenty-five years, and attributed his condition to malaria. A diagnosis had been made by the physician of rheumatoid arthritis, or possibly he really was suffering from true gout, as he was intemperate. No sooner was it insisted upon that he should be regularly attended to for his stricture than the "rheumatism" disappeared and he had little to complain of than his bronchitis.

(6) A patient, æt. 50 years, of a highly nervous temperament and very spare bodily development, came under observation for recent enlargement of each epididymis. He had contracted syphilis sixteen years previously, but had been most radically treated and had apparently never suffered from any secondary or tertiary signs. Two sisters had died of consumption. As masses had developed in each epididymis and as his general health was so impaired he had been advised to break off his engagement to marry. The temperature was normal; the lungs presented no signs of tuberculosis, and four tuberculo-opsonic observations made shortly after exercise were found to be within normal limits. He was also subject to difficulty in passing water and was under treatment for stricture of the urethra. A sample of urine was examined; it was highly offensive, and though at first the odour suggested ammoniacal decomposition, yet more careful investigation showed that there was no free ammonia present (and the sample was quite acid to litmus paper), and that the odour was more that of decomposed fish. The urine

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was free from coarse deposits of pus, though a few particles were noticed which were composed of pus cells and fibrin. The urine on standing showed a slight tendency to form a deposit, but only a small upper portion became quite clear. The "mucus" on microscopic examination was found to consist of a few pus cells, small amorphous particles and countless bacteria, which proved to be the coli-bacillus. No tubercle bacilli were found. On ordinary clinical examination this case, by reason of the wasting and general debility and the deposits in the epididymis gave every reason for the suspicion of tuberculosis, and the family history confirmed this view. It may be anticipated that, like similar cases already described, this one, after regular attention to the obstruction in the urethra caused by strictures which had been neglected, would be freed from much of the mimicry of such a disease as tuberculosis.

(7) A man, æt. 47 years, had had symptoms on and off during the previous six years from attacks of dyspepsia lasting two to three weeks. His general appearance was excellent, despite the fact that he habitually slept only five hours out of the twenty-four, and had an indifferent appetite. He was inclined to be morose, and inveighed bitterly against the medical profession in Buenos Ayres, Paris and Carlsbad. He had been dieted to remedy hypochlorhydria and possibly duodenal ulcer, had had repeated gastric analyses carried out, and was hotly opposed to having any further lavage of the stomach by means of a stomach-tube. He had had gonorrhœa and syphilis and there was a scar in the right groin.

The condition of the water in this case was almost identical with the last, with the added feature that the sample of urine sent for examination was so highly gaseous that the cork was driven from the bottle after standing in a warm place. Methods designed to relieve the stricture and to irrigate the bladder were followed by a complete restoration to health, although the urine, which had become much clearer, still contained the coli-bacillus. It is difficult to say how much the improvement in the symptoms of dyspepsia was due to the reduction of the coli-bacilluria, and how much to the discovery that the urinary tract was the seat of an infection which responded to treatment.

(8) Through the courtesy of Dr. Wethered reference is now made to the notes of a girl, æt. 14 years and 10 months, who was admitted to the Brompton Hospital for pulmonary and peritoneal tuberculosis. Previous to admission the bladder was found to be distended and the patient had been unable to pass water; a catheter was passed and the bladder emptied. Three days later the patient passed water which was "ammoniacal" (?) and contained blood, pus phosphates and oxalates, and coli-bacilli in pure culture; the specimen was the first passed since the catheterisation. The patient died eight days after this operation had been performed. Dr. Inman, the Superintendent of the Pathological Laboratory of the Brompton Hospital, discovered at the autopsy that the bladder was the seat of a most intense, hæmorrhagic, croupous inflammation, the false membrane covering the whole interior of the bladder. The ureters were quite normal except at their junction with the bladder, where they were slightly hyperæmic; there was no evidence of suppuration of the kidneys; coli-bacilli were found in the false membrane and bladder wall. Besides tuberculosis of the peritoneum and lungs tuberculous meningitis was also detected. Though not absolutely proved, as shown by the history and course of the disease, it it likely that this patient developed a toxic suppression of urine and inflammation of the bladder as a result of the passage of the catheter and infection with the coli-bacillus.

(9) A married woman had suffered from pains in the lower abdomen for three weeks following upon an attack of severe leucorrhœa lasting three weeks; there was difficulty, pain, and frequency of micturition. She was admitted for pelvic peritonitis, but as the pelvis was found to be normal and no other cause found the Widal test for typhoid fever was carried out as the temperature chart was suggestive. Agglutination occurred with dilutions of 1 in 30 and 1 in 50, but not with a dilution of 1 in 100. The urine was found to be slightly purulent and was loaded with "mucus," which proved to be largely composed of the coli-bacillus; the symptoms and temperature gradually abated, and the pyuria was almost cured by instillation of the bladder with a solution of 1 in 1000 silver nitrate.

(10) A lady, æt. 64 years, otherwise very healthy, had for several years been liable to attacks of "cystitis," *i. e.* there was great frequency of micturition,

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and considerable strangury, and pus appeared in the urine, which was for a time highly offensive. In the intervals between the attacks the urine was quite normal. No explanation could be found for the recurrence of these attacks; stone in the bladder was excluded by sounding and there was no suspicion of tuberculosis. A bacteriological examination of the urine of this patient has not been possible, but from its similarity to other cases of "recurrent cystitis," in which this examination has been effected, it may be surmised that like them the recurrent attacks of cystitis were due to recurrent coli-bacilluria.

(11) A young single woman who had always been healthy, with the exception of occasional pains in the left loin, was suddenly seized with rigors and severe prostration accompanied by fever which reached 104° and 105° F. The urine was soon noticed to form a deposit, at first mistaken for phosphates, but eventually it was proved that the deposit was in the main due to pus cells. Rather severe symptoms of cystitis developed and gas passed with the urine (pneumaturia). The urine, on closer examination, was found to contain numerous coli-bacilli; tubercle bacilli were absent and the results of radioscopic examination were negative. It is obvious that some connection had been established between a urinary tract, presumably that belonging to the left kidney, and the bowel, and that coli-infection had taken place, gas developing in the bowel being discharged by the urinary tract.

Comment.—Little purpose would be served by giving details of other cases which were proved to be due to, or were suggestive of, infection of the urinary tract. In this short series sufficient evidence is brought forward to show that infection of the urinary tract, and more especially infection by the coli-bacillus, is capable of producing or being associated with the similitudes of malaria, dysentery or mucous colitis, recurrent cystitis of unknown cause, enuresis, pyuria of unexplainable cause, "gout" or "goutiness" or "rheumatism," general tuberculosis or genital tuberculosis, dyspepsia (? duodenal ulcer), acute suppression of urine. Other cases could be quoted in which the symptoms and signs were simulative of quite different forms of disease, such as typhoid fever and pernicious anæmia. No doubt the condition is familiar enough to clinicians, but it is equally true that the importance of the infection is hardly sufficiently recognised except in those cases in which pus, by reason of its large volume, attracts attention. Blumenthal (1) even as late as 1903 points out that "bacteriuria occurs in cystitis and pyelonephritis, producing a diffuse but uniform turbidity of the urine," but a discussion of the flora of the urine in pathological conditions of the urine receives from him but scant attention, at least in those cases in which pyuria is not a gross feature.

Pathological Considerations.

The more common organisms which cause infection of the urinary tract.—Investigation has shown that various micro-organisms are capable of gaining access to the urinary tract, and after once gaining access experience shows that it is very difficult and even impossible to secure their complete removal. Various figures are quoted as representing the more frequent infections. Faltin (2) was able to show that the coli-bacillus headed the list with a proportion of 42–43 per cent.; then follow staphylococcic infection 25 per cent., and streptococcic infection 20 per cent. Other organisms found to be present were the *Bacillus pyocyaneus*, the *Bacillus proteus*, and various putrefactive organisms; not only aërobic organisms, but even anaërobic organisms appear also to be able to flourish.

Escherich (3) has investigated sixty children, in whom the infection had assumed such clinical characters as to justify the use of the term "coli-cystitis"; in fifty-eight of these cases coli-bacilli were found to be present. Rostoski (4) investigated the nature of the infection in 120 adults, and found the *Bacillus lactis ærogenes* (an organism which is closely similar to the coli-bacillus) in no less than eighty of the cases.

It is obvious, therefore, that the most common infection of the urinary tract is by the coli-bacillus, and a possible explanation of the comparatively small importance of this infection is that though by far the most common infection it is certainly not the most dangerous.

Staphylococci and streptococci are capable of producing a much more grave disturbance of the system, and therefore are much more likely to attract attention [Rosving (5)]. Some of the gravest examples of infection of the urinary tract have been shown to be due to the joint action

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of the coli-bacillus and staphylococcus or streptococcus.

Nomenclature adopted. — In the monographs dealing with the infection of the urinary tract, viz. those of Faltin (6), R. Barlow (7), and Viktor Blum (8), considerable information can be found as to the nomenclature which has been adopted. No doubt now that so much more is known of the specific clinical characters of various infections and of the nature of the protective defences set up by the body capable of being determined in the laboratory, much of the earlier difficulty of finding a term which should aptly describe infection of the urinary tract has been dissipated. It is a matter of national gratification to know that interest in the subject of urinary infection was first aroused as the result of a discussion in 1883, before the Medical Society of London, in which the late Sir Andrew Clarke, Sir Joseph Fayrer, Sir Henry Thompson, Mr. Berkeley Hill, and Mr. Marcus Beck took part, and it is of still greater satisfaction to know that one of the best names for the description of this group of disorder was introduced by the late Sir William Roberts. Although it was left for later workers to prove that the growth of organisms in the urinary tract was capable of producing signs of systemic poisoning, Sir William Roberts was the first to introduce, in 1881, the term "bacteruria" to describe a condition in which the sediment of the fresh urine on microscopic examination was found to consist largely of rod-shaped organisms. Other terms which have been used are "bacteriuria," "urosepsis," "urinary infection," etc. It has been maintained that the term "bacilluria" or "bacteriuria" should be restricted to those cases in which, although organisms are present, they have failed to produce symptoms and have not been provocative of pyuria, and that "uro-sepsis" and "urinary infection" should only be used as descriptive terms when these bounds have been overstepped to the extent of producing sapræmia and septicæmia and coarse signs of pyuria. That such an artificial separation of infection is harmful to the due interpretation of the gravity of urinary infection is shown by the short clinical accounts already referred to. Because obvious pus was not observed in the urine by naked-eye examination in several of the cases, cardinal errors were made in the diagnosis, and it is a familiar experience that when urines are subjected to centrifugalisation, pus-cells are found abundantly when previously their existence was negatived. Cerebro-spinal fluid, if not previously centrifugalised would most certainly be passed as free from cellular contents in certain pathological conditions. Moreover, the existence of such terms as "catheter fever" (Andrew Clarke), "urinary fever" (Henry Thompson), "urethral fever," "malaria-occulta" (Joseph Fayrer), "uræmic fever" (Richeraud), "urinary poisoning" (Guyon, Malherbe and other French writers), and "urinæmia" (Posner) shows how little the true cause of many of the symptoms following catheterisation was appreciated when first the subject began to be studied. Despite the failure experienced by Bouchard and his followers to convince others that such symptoms as follow catheterisation might be due to the toxic effects of urinary excreta, apart from the action of bacteria, Blum maintains that some at least of the cases in which constitutional disturbance follows the use of the catheter, more especially those in which the symptoms follow within a few hours of instrumental interference, are most aptly described as "urotoxæmia," reserving the term "urosepsis" for that group of disturbances which are certainly due to infection of the urinary tract. That it is possible to still further proceed to greater refinement is shown by the advances made in the study of immunity, for by these means it is possible to show that although ordinary clinical methods fail to show any sign of infection, investigations on the agglutinative and other properties of the blood of patients, whose urinary tract is the seat of infection, are able to reveal in the same subjects distinct evidence of systemic intoxication, and as a result of blood cultures even of systemic infection. The general result of these advances has been to disregard the artificial distinctions hitherto adopted between bacilluria and pyuria, and by special methods of a bacteriological order to endeavour to substantiate a typho-bacilluria, coli-bacilluria, tubercle-bacilluria, etc.

Factors influencing the severity of the symptoms originated by urinary infection.—It has already been stated that various pathogenic organisms may be present in the urinary tract without in the least degree affecting the health of the individual as noticed by himself or even by the physician. The first sign to call attention has been usually that the

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urine may have been found to contain a whitish deposit, and this has been found to be pus containing various pathogenic organisms; occasionally, as in coli-bacilluria, it is the extremely offensive character of the urine when passed that has drawn attention to possible disturbance of the urinary function; typho-bacilluria may be, and often is, but an interesting clinical phenomena, so far from complicating convalescence from typhoid fever, actually not requiring any special treatment for its complete spontaneous subsidence. Such cases form further examples of what has recently been described as "typhoid carriers," as opposed to true typhoid patients. The ground, however, is very seriously altered whenever obstruction occurs to the complete emptying of the bladder, and as these cases are just those in which instrumental aid is required—stricture of the urethra, prostatic enlargement, bladder and prostatic neoplasms, pregnancy, and other tumours of the pelvic viscera obstructing the escape of the urine in a normal way—so it happens that these cases have been more carefully studied by the surgeons and gynæcologists than by the physician. Not in all cases, however, are such obstructions found to exist, and yet the patient suffers, without doubt, from symptoms of true urosepticæmia or uro-sapræmia, and it is to one of this particular group of cases (cases which might well be spoken of as *Bacillus coli* carriers) that this contribution is designed to call attention. There can be no doubt that an individual may harbour in the urinary tract pathogenic organisms, quite as harmful (for a time) as those which infest the respiratory and intestinal tracts, and be free from symptoms, and yet as soon as the natural defences of the body break down, either from a depression of the immunising powers of the body or from an exaltation of the pathogenic powers of the organisms found, symptoms of general intoxication, and even of general infection, at once occur. Moreover, it is quite possible, and clinical impressions support the view, that organisms possessing the same degree of virulence when introduced into the bladder of different individuals possessed of different powers of resisting infection and intoxication, whether by means of age or of previous debilitating illness, can and do produce effects which present very varying and corresponding degrees of severity. Finally, all causes which lead to an imperfect flushing of the urinary tract by the natural secretion are greatly operative in increasing the possibilities for general infection and intoxication. The detrusor action of the bladder is dependent upon a healthy condition of the nervous mechanism which subserves micturition upon a bladder wall which is readily responsive to such nervous control. It is little to be wondered at that catheterisation, which in a healthy individual is followed, not only by no symptoms but by no chronic infection of the urinary system, in part or whole, would be fraught with much danger in an individual suffering from grave disturbances of the central or peripheral nervous system. The grave intoxication which follows in the immediate wake of instrumental interference with the urinary tract may take the form of somnolence and such intense apathy to the natural calls for micturition that the intoxication is still further intensified by a retention within the bladder of organisms and even of their products, which under more favourable conditions would be readily evacuated. Urosepticæmia and urosapræmia is much more marked and more frequent when the site of the infection is mainly the bladder rather than when the renal pelvis, the ureters, or the urethra are alone affected; probably this is dependent purely upon consideration of extent of surface through which absorption can take place. Probably also under healthy conditions no part of the surface of the urinary system absorbs any of the soluble substances evacuated by the kidney. Whenever retention of urine occurs and the pressure is greatly raised, absorption takes place as a result of the internal disorganisation of the urinary surfaces. These internal disturbances are the direct result of the action of retained bacteria and their products; mechanical causes such as follow instrumental interference also operate in the same way. It seems to be clearly proved that of all parts of the urinary system, those most likely to yield to the above injuries are the pelvis of the kidney, the bladder and the prostatic part of the urethra.

The Mode of Origin of Coli-Bacilluria.

The coli bacillus may gain access to the uropoietic system by being introduced by infected instruments. The consideration of this subject is hardly one which concerns the physician, at least in the first instance. So comparatively

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simple is it by the adoption of various antiseptic and aseptic procedures to secure the sterility of the instruments used that such a source of infection needs no consideration here. The same may be said with respect to the adequate toilet of the genitalia before instrumentation. It is, however, a matter of grave importance to consider whether it is possible to avoid pushing into the bladder organisms which are already resident in the urethra; despite all efforts to secure asepsis of the urethra before introduction of instruments, it is difficult to believe that it is possible in every case to insure that such an accident should not occur. The anatomical configuration of the lining wall of the urethra is so inimical to complete sterilisation that given the presence in the urethra (especially of the male) of the coli-bacillus, all the preliminary lavage of the urethra that is possible can hardly be held to be sufficient for such purposes. That the coli-bacillus is a common denizen of the urethra is shown by the observation of Melchior (9), who found it present in the meatus urinarius in 50 per cent. of all cases examined.

Not only is it not sufficiently recognised that the coli-bacillus is a common inhabitant of the urethral channel, but it is also inadequately understood that the coli-bacillus gains access to the uropoietic system from within the body. It is well known that coli-bacilluria may occur in subjects who have never been subjected to catheterisation. A ready suggestion has been made that under various circumstances in which the urethra is not frequently flushed from above by the natural secretions of the urinary system, that the coli-bacillus ascends along the urethra by a process of colonisation or spontaneous growth until it reaches the bladder, and by a similar process may gain access to the kidneys. A repetition of this form of infection may occur, more especially in women and girls, in whom spontaneous coli-cystitis is more common than in males. No doubt the circumstantial evidence in favour of this view hardly carries complete conviction, and various writers have endeavoured to find other sources of "internal" infection by the coli-bacillus. It is believed that any injury to the rectum is capable of admitting to the submucous tissues the coli-bacilli which normally inhabit the bowel; the organisms are those picked up by the lymphatic vessels and transmitted to the adjacent bladder wall, there to gain access to its interior. Wreden (10) has shown that if the wall of the rectum is experimentally injured short of establishing a fistula into the bladder, subsequent injection of oil or vaseline into the rectum will be followed by the appearance of these substances in the bladder, and it is on record that the cure of an anal fissure has been followed by the disappearance from the urine of coli-bacilli previously present. The occurrence of typho-bacilluria during the course of, and convalescense from, typhoid fever further supports this view. Trumpp (11) has also shown that coli-bacilluria occurred in fourteen children out of a total of seventeen who suffered from follicular enteritis. Moreover it is possible that when the coli-bacilli have invaded the system generally they are partly extruded by the kidneys and so gain access to the bladder. It may be urged that as the coli-bacillus is such a common denizen of the alimentary tract, its transference to the bladder is hardly likely to produce any constitutional disturbance, seeing that already it has had such admirable opportunities to do harm by means of its occurrence in the intestine. The interesting observation has been made by Pfaundler (12), however, that although the coli-bacillus is a natural inhabitant of the bowel, it does not as a rule appear in these circumstances to lead to the development of any agglutinative action of the serum of the normal individual. As soon, however, as the coli-bacillus gains access to the bladder or kidney pelvis, agglutinins develop, so that coli-cystitis set up by the presence in the bladder of coli-bacilli ordinarily present in the adjacent rectum is capable of being accompanied by a Widal reaction against the coli-bacillus.

The Bio-chemical and Toxic Characters of the Colon Bacillus in relation to Coli-Bacilluria.

(1) *Bio-chemistry.*—There can be little doubt that the reason why, from a clinical point of view, coli-bacilluria has failed to attract its proper measure of attention, is that a urine which fails to give a reaction for proteid or sugar, is of normal specific gravity, *and is acid*, is usually passed as normal, any increased turbidity being only too frequently ascribed to a physiological increase of mucus derived from the disintegration of the lining of the urinary passages. So strong a hold has the knowledge that certain organisms such as the *Micrococcus*

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ureæ and even the *Staphylococcus pyogenes* and others produce an ammoniacal decomposition of the urine, that it has been too readily accepted that the coli-bacillus, if present in the urine, would also cause ammonia to be formed and lead to an alkaline reaction. In view of the contradictory opinions as to nature of the changes brought about when the coli-bacillus is sown in sterile urine or in solutions of urea, such a view has some excuse. The normal urine is usually considered to be free from sugar and from the hydrolytic products of proteid digestion: occasionally peptone and lactose may occur under apparently physiological conditions, but not with a constancy which would require that such bodies should be considered as capable of providing means for the known activity of coli-bacilli when given upon media containing sugars or proteid derivatives. It is thoroughly well established that the coli-bacillus is capable of growing in media containing grape sugar or peptone, both in aërobic and anaërobic conditions, and that generally speaking when grown on media containing grape sugar various acids and gases are formed, and when grown on media containing peptone ammonia is formed. But these bodies do not occur in normal urine so that when the coli-bacillus gains access to the urine, there is no reason to believe that the reaction should be altered either in the direction of increased or decreased acidity or alkalinity; coli-cystitis, as shown by Escherich, is characterised as an acid cystitis. Repeated experimental observations have been made, all of which show that the coli-bacillus when grown in sterile urine or urea solution does not give rise to the formation of ammonia or carbonic acid by the destruction of the urea, or only to a very slight extent, either because such a medium is unsuited for growth of the organism or because the organism has lost the power of cleaving amido-bodies. Although the majority of observers support this view, the opposite is held by some workers, and one of the tests by which the coli-bacillus is separated from the typhoid bacillus turns on the observation that the former organism, when grown on a medium consisting of agar, lactose and urea, first develops an acid and then an ammoniacal reaction. That this cannot be due, however, to a conversion by the coli-bacillus of urea into ammonia is shown by Escherich (15), who, although able to show an increase of ammonia when the coli-bacillus is given in a medium containing urea, was unable to demonstrate a decrease of the urea: possibly the ammonia formation under these circumstances is due to the intra-cellular enzyme action of the bacilli upon the nitrogenous constituents of the bacillary body. Despite the want of agreement of the various experimental observations, the clinical fact remains that the urine in coli-bacilluria does not become alkaline; it remains acid.

Another point upon which attention must be directed is that the urine of patients suffering from coli-bacilluria may have an offensive smell quite different to that of normal urine, and sufficiently unpleasant to even attract the patient's attention. Normal urine does not contain any native proteids, so that the coli-bacilli cannot form offensive substances from them, and it is a fact that even were they present the coli-bacilli would be quite incapable of leading to the decomposition of such bodies as they are non-proteolytic. Should, however, any proteid derivatives occur in the urine or albuminoid substances, then the coli-bacillus is capable of leading to their further disintegration, and possibly they could give rise to various odorous substances. It is known that abscesses containing pure cultures of the coli-bacillus yield offensive gases which suggest the presence of methyl-mercaptan, and, moreover, the coli-bacillus can form pure sugar-free media, such as gelatine, agar-peptone broth, another offensive gas, namely, sulphuretted hydrogen. It is therefore possible, especially under the pathological condition evidenced in the urinary tract by the presence of the coli bacillus, that the characteristically offensive odour of coli-bacilluria occasionly met with is due to admixture of the ordinary urinary odour with methyl-mercaptan and sulphuretted hydrogen; ammonia, however, plays no contributory part.

(2) *The toxic properties of the coli-bacillus.*—Possibly one of the great reasons why the coli-bacillus occurring in the urine has not attracted the attention of clinicians as a possible source of various symptoms, is the fact that bacteriologists are not in agreement that it is capable of producing toxic substances when grown on suitable media, *i. e.* that there are no exotoxines. Celli (13) and Alessandri (14) allege that a variety of coli-bacillus recovered from some dysenteric evacuations, when grown on bouillon, produced toxic substances which,

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when precipitated by alcohol, yielded a toxine which when injected into carnivora produced dysentery, wasting, and fever. Escherich (15) was, however, unable to confirm these findings. Even were such toxines capable of being formed within the human bladder infected with the coli-bacillus, it is probable that the normal evacuation of urine would prevent the absorption of such toxines so long as the bladder wall remained healthy. As already noted, the existence of a damaged bladder wall or of a real or virtual obstruction to the escape of urine would render such absorption more possible.

Greater unanimity of opinion is, however, met with as to the existence of endotoxines. These bodies actually exist, but though on absorption or introduction into the system they provoke the formation of bactericidal, agglutinating, and precipitating anti-bodies, so far it does not appear that anti-toxines are formed.

When once the coli-bacillus disintegrates then endotoxines may be liberated. Buchner found that pyogenic material could be obtained from coli-cultures which had been killed by heat. And Escherich and Pfaundler quote the observations of Gilbert, who found that the intra-cellular metabolites of the coli-bacillus when introduced into the circulation are capable of producing paralysis of voluntary muscles, loss of sensibility, coma, convulsions, increase of reflexes, and even death. Asch (16) has also shown that the death may be rapid, occurring in a few hours or days, and that even chronic nephritis may be initiated. It is thus clear that experimental evidence has shown that endotoxines may be produced when the coli-bacillus undergoes an artificial disintegration. It is frequently seen on the examination of the urine in coli-bacilluria that there is an admixture of motile bacilli with degenerated and non-motile individuals, and it may well be believed that the latter are yielding to the urine definite toxic products, ready to be absorbed into the system whenever, by reason of defective evacuation, the urine is allowed to be retained for a considerable time in the bladder.

The urine in coli-bacilluria.—A more careful examination of the urine is necessary than the ordinary routine one. Reference has already been made to the offensive odour, though this characteristic is not always noticeable. Moreover, the urine is acid. Probably oversight of the true condition of the urine is first begun when a turbidity of the urine is interpreted as merely an excess of the normal mucus. Coli-bacilluria is also accompanied by an increase of the natural amount of mucus, consequent upon the mild inflammation of the urinary passage. This mistake is the more likely, because after standing the diffuse turbidity may in certain cases assume the form of a deposit, though in more marked cases this is never so complete as to mislead the cautious observer; moreover, the turbidity is much denser than when mucus alone is responsible for the want of clearness of the specimen. The observer may be tempted to suspect urates or phosphates, but the usual reactions for these substances fail. A common feature to be noticed is that heat and acetic acid do not lead to the clearing of the solution, in fact there may be a slightly increased turbidity of that part of the urine submitted to the test; so slight indeed is this, however, that it may be overlooked. Prolonged treatment with liquor potassæ instead of leading to a complete clarification of the specimen produces only a partial effect, small cloudy formations developing, which gradually sink to the lower end of the tube. Occasionally the turbidity, instead of being a diffuse haze, takes the form of minute flocculi, which remain suspended evenly throughout the whole depth of the urine-glass.

A microscopic examination of a drop of the urine reveals countless motile bacilli, sometimes so short as to resemble cocci; at others long, thread-like individuals may be seen. By the use of Loeffler's methylene-blue the organism is readily made more visible. It is wise also to stain a film of the urine by Gram's method, as by this means mixed infection with various staphylo- and streptococci may also be established. As already remarked, pus may be so small in amount as only to be detected by microscopic means.

Symptoms.—It will be frequently found that patients (especially women and girls) are entirely free from any symptoms whatever; the discovery of coli-bacilluria is therefore made in these cases entirely as a result of routine examination of the urine.

Sometimes, however, as in one of the cases recorded, there may be frequency of micturition with or without pain, limited frequently to the day only, or occurring both day and night, an important matter, as coli-bacilluria may be an explana-

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tion of many intractable cases of enuresis—*e. g.* in children, and may be an explanation for those cases of enuresis developing in adult age, and which at once arouse a suspicion of involvement of the spinal cord in diseases as, *e. g.* in tabes dorsalis.

It happens sometimes that the patient notices that the urine, when voided or allowed to stand a few hours, is very offensive. Or the only symptom may be an uncontrollable irritation of the genitalia; in some cases there is distinct pain associated with the act of micturition. It is only necessary to remind you that there may be grave constitutional disturbance, even to suppression of the urine when the infection has spread along the urinary passages to the kidneys.

If the bladder wall is quite healthy and there is no obstruction to the outlet of urine general symptoms do not arise, for absorption from the bladder of toxines is under usual conditions impossible. Should this occur, however, then a probable explanation is found for the association with coli-bacilluria of indisposition, fever, rigors, and even enlargement of the spleen, possibly also of dyspeptic symptoms, odd rheumatic pains, mucous proctitis, etc., and drowsiness, sickness, nausea, loss of appetite, and even diarrhœa and constipation.

Viktor Blum, and R. Barlow have given an excellent summary of the various clinical types of the disease, and they fall into one or other of the three following groups:

(1) *Symptoms mainly characterised by fever of an apparently unexplained origin.*—The attacks of fever may be very acute, and may be brought about as the result of catheterisation or independently of this operation. They may last but a day or so and recur with such regularity as to justify the opinion that the patient is suffering from malaria. In cases in which the febrile reaction assumes pyæmic characters other organisms, such as the staphylococcus or streptococcus, may be suspected as mixed infection. The coli-bacillus has been actually recovered from the blood during the febrile attacks. The patient may present signs of valvular infection and so present an appearance of malignant endocarditis. Lenhartz (16) found that out of thirty-eight cases of fatal malignant endocarditis seven dated from the damage done to the urethra by the use of catheters, bougies, etc.; the coli-bacillus has been recovered from the valves of cases of fatal endocarditis following upon examination and treatment for stricture.

(2) *Gastro-intestinal symptoms.*—In one of the cases already quoted the symptoms were very suggestive of hyperchlorhydria and even of duodenal ulcer. It is also known that gastric or intestinal carcinoma may be closely simulated. The symptoms may be acute or chronic and include occasional sickness, nausea, loss of appetite, headache, diarrhœa and constipation, and the causal connection of these symptoms with the coli-bacilluria is shown by their disappearance under treatment directed against the infection. In the more chronic cases cachexia may be a marked feature; the skin even may be so yellow as to suggest jaundice; the sclerotics are, however, free from any coloration. In other cases the patient is so pallid as to suggest the existence of pernicious anæmia. The mouth is often found to be dry, necessitating frequent drinking of water; even water brash and bitterness of the oral secretion occurs. A sensation of pressure or of pain may occur immediately or shortly after taking food or drink; even hæmatemesis may accompany the vomiting.

(3) A final group includes the cases showing *cerebral* symptoms. In the milder cases only headache and giddiness are noticed, or even migraine and tinnitus aurium. The more severe cases simulate uræmia; a marked desire to sleep may be a feature.

No doubt the severer forms of these clinical types would suggest the existence of consecutive nephritis, and when the bladder symptoms are severe, coli-cystitis would be suspected and looked for; but it is necessary to once more point out that frequency of micturition and pyuria of well-marked type may be quite absent, and the urine present none but the slightest evidence of departure from the normal (if a bacteriological examination is not included), and yet all the symptoms belonging to one or other of the above three clinical pictures be present, dependent entirely upon coli-bacilluria. Moreover, when it is remembered that frequently patients enjoy periods of excellent health between the attacks, and the urine may assume an entirely normal character, the difficulty of diagnosis without a methodical examination of the urine, especially during the attack, is at once apparent.

Prognosis.—Unfortunately, when once coli-bacil-

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luria has been developed, it is most difficult indeed, in some cases absolutely impossible, to effect a cure. Some cases, however, undergo a spontaneous cure; in others remissions occur, the patient in the interval being entirely freed from symptoms.

Treatment.—There can be little doubt that increased personal cleanliness would lead to a reduction of the number of cases of coli-bacilluria developing independently of the use of catheters, bougies, etc. Moreover, if the practice of passing a catheter when symptoms first develop of enlargement of the prostate in man were less resorted to, the grave forms of coli-bacilluria would be reduced in number. There is little doubt that of all the urinary disinfectants given by the month, urotropin and its allies (helmitol, etc.), are the most popular, but frequent use of this drug for such purposes has carried conviction to most observers that although it is an admirable means of allaying the sensation of irritability of the bladder, its bactericidal action falls far short of that which reputation gave it when first introduced.

Probably the best remedy for cases of coli-bacilluria developing independently of instrumental interference is to leave them alone, unless the symptoms are such as to call for some palliation; other cases of urinary infection may go on for years and yet not lead to any serious symptoms. Finlayson (18) described a case of infection of the bladder with sarcinæ which had lasted for fifteen years. If it is suspected that for various reasons the bladder is not completely evacuated then the surgical measures usually adopted for such cases should be rigorously carried out. If coli-bacilluria is dependent upon an infection of the bladder only, then no harm will follow and much good may result, as judged by the experience of most observers, from the daily lavage of the bladder with a solution of silver nitrate of the strength of 1 in 1000 to 1 in 2000 mm., even 1 per cent. solution in severe cases, may be necessary. Seeing that it has not been established that the endotoxines of the coli-bacillus can provoke an anti-toxine formation serum-therapeutics may be anticipated to be a failure, a result which a few attempts in this direction have proved to be the case. Excellent results have, however, been achieved by vaccination, but even this therapeutic measure can scarcely be successful if the infection has passed beyond the limits of the mere surface of the urinary tract, an effect which is much the more likely when obstruction to the complete evacuation is effected by the existence of an enlargement of the prostate, strictures, tumour, etc.

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February 10th, 1908.

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CASE OF FATAL ACUTE ILLNESS IN A CHILD FROM "STATUS LYMPHATICUS."*

By SIDNEY PHILLIPS, M.D., F.R.C.P.,
Physician to St. Mary's Hospital.

W. P—, a boy, æt. 5½ years, was stated by his mother to have always had good health, and to have been quite free from any sign of illness until October 12th, 1906, when his breathing began to be noisy and difficult. He was at once taken to a doctor and afterwards remained under medical treatment at home, though not in bed, as his breathing became worse in the recumbent position; probably for this reason it was worst at night, sometimes so bad that he rose up screaming. The breathlessness increased day by day, and he came into St. Mary's Hospital on October 15th.

I saw him the same afternoon. He was a well-developed, well-nourished boy, propped up in bed, gasping for breath in great distress. The respirations were 36 a minute, and with each inspiration there were three sounds very like a hiccough thrice repeated; the first of the three sounds was louder than the other two, which were separated from one another by a shorter interval than from the first sound. The effect is best represented by uttering the sounds "hick—heck-heck." There was contraction of the platysma muscles and drawing downwards of the lower jaw with each inspiration, and marked sinking in of the epigastric and supraclavicular regions. The boy was too breathless to speak much, but when he shrieked out, as he did at times in his extreme distress, the voice was quite clear. He was pale rather than cyanotic and his extremities were not cold; the pulse was feeble and small, the temperature normal; at times he came out in a clammy sweat. There was moderate chronic enlargement of the tonsils. I could find nothing abnormal in the larynx, nor could my colleague, Dr. Scanes Spicer, who kindly examined him; the vocal cords moved freely.

There was nothing wrong detectable in the lungs, though the breath sounds were weak over both sides of the chest.

Dr. Simmonds, at my request, screened him with Röntgen rays, but there was no sign of any foreign body in the air-passages.

He had been sent into hospital for diphtheria, but there were no signs of membrane anywhere. It was clear the breathlessness was not due to laryngeal or to pulmonary disease. The peculiar character and sounds of the breathing were much like those sometimes observed in hysterical girls, but the condition here was much too grave to attribute merely to a neurosis.

There was a suspicion of some want of percussion resonance over the sternum, and I concluded that the dyspnœa probably arose from pressure on the air-passages in the mediastinum. Not at the time thinking of the thymus gland, I thought such pressure probably arose from enlarged lymphatic glands about the tracheal bifurcation, or even their ulceration into a bronchus as in the case recorded by Mr. R. W. Parker in the 'Clinical Society's Transactions' (vol. xxiv). Tracheotomy in such a case could be of no avail, and all that could be done was to administer a little morphia, which gave some temporary relief. I saw him again at 10 p.m., but the distress was as great as ever and the pulse weaker. He died during the night, apparently from heart failure.

The autopsy was made next day by Dr. Spilsbury, and nothing was found wrong in any organ of the body, except an enlargement of the thymus gland and certain changes in the spleen and lymphatic glands which are described by Dr. Spilsbury as follows:

Thymus.—Weight, ¾ oz.

The thymus consisted of two lateral lobes closely opposed, but not united; it extended from the lower border of the thyroid body downwards into the mediastinum, its lower part coming into close relationship with the pericardium. The surface of the organ was lobulated and on section it was fairly firm.

Histology.—*Thymus* showed hyperplasia of the lymphoid tissue, which extended outwards into the neighbouring areolar tissue and inwards into the medulla of the lobules. The medulla and the cortex were therefore indistinctly marked off from each other.

Coarsely granular eosinophilous leucocytes were present in large numbers.

Hassell's corpuscles were numerous and large.

Mesenteric lymph-gland showed simple hyperplasia. The lymph-nodes were large and in some places two were fused together. The gland was well supplied with blood-vessels.

* Delivered before the Royal Society of Medicine.

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Spleen showed enlargement of the Malpighian bodies due to hyperplasia. The spleen pulp was extremely congested and showed considerable deposit of pigment in the form of fine granules, both inside the cells of the spleen pulp and lying free.

Liver showed early nutmeg congestion and slight fatty degeneration. There seemed to be no accumulation of pigment in this organ.

Remarks.—The case seems to be an example of the affection at one time spoken of as "thymic asthma," and more recently as "lymphatism" or the "status lymphaticus."

Cases of unexpected death in young infants, apparently from cardiac failure after attacks of dyspnœa with or without convulsions, in which no abnormality has been found post mortem except enlargement of the thymus and lymphatic glands, have been frequently recorded.

Grawitz ('Deutsche med. Wochenschrift,' 1888, vol. xiv, p. 429) records the case of a child, æt. 8 months, found dead in bed without any previous sign of illness, and a second case of a child, æt. 6 months, which died in its father's arms after a few moments of illness; in each of these cases the thymus gland was found greatly enlarged post mortem, and in the second of the cases there was also enlargement of spleen and mesenteric glands.

Jacobi ('Trans. Assoc. American Physicians,' vol. viii, 1888) records the sudden death of a child, æt. 6 months, which after autopsy he could only attribute to enlargement of the thymus gland.

Dr. Crozier Griffith, in the same publication for the year 1903, gives notes of a case of a child, æt. 7 months, and of a child, æt. 6 months, both of whom died suddenly, apparently of syncope, after sudden difficulty in breathing.

Each of these children had had convulsions, though not at the time of the fatal illness.

Instances in which sudden death with dyspnœal attacks has been found associated with enlargement of the thymus and of the lymphatic glands are so numerous that it has been concluded that there is more than mere coincidence.

Death in these cases has been attributed to larnyngo-spasm, but laryngismus stridulus does not, I think, kill off infants in this sudden way, certainly not a child of the age of my patient, and in none of the recorded cases is there any mention of the characteristic crowing breathing; death, too, has appeared always to have resulted from cardiac failure.

The theory of laryngo-spasm appears to have been put forward in default of any better explanation at a time before the changes had been observed in the thymus and lymphatic glands.

Death has been attributed to pressure upon the trachea by the enlarged thymus gland. Morfans ('Soc. Med. des Hopitaux,' 1894, p. 361) found the trachea flattened and its calibre narrowed in an infant, æt. 2½ months, which died with an enlarged thymus gland. Koenig ('Central. für Chirurg.,' 1897, p. 605), Perrucke ('Gaz. Hebd. de Med. et de Chirurg.,' 1889, p. 695), and Siegle have each recorded a case in which after the enlarged thymus gland had been raised from over the trachea by operation the symptoms were relieved.

These cases go to disprove Friedleben's statement that it is impossible for the trachea to be compressed by the thymus gland. Nevertheless pressure is not the cause of the dyspnœa in the majority of the cases, for in most of them the thymus gland is but slightly enlarged and no signs of pressure upon the trachea are found. And, as Crozier Griffith remarks, if the symptoms arose from pressure they would not come on so suddenly as they do.

Paltauf ('Wien. klin. Wochenschr.,' No. 46, 1889, and No. 9, 1898) advanced the theory that the enlargement of the thymus and lymphatic glands of the body were evidences of a constitutional state in which sudden cessation of the heart's action may occur from very slight causes—among others administration of anæsthetics. This theory is supported by Escherich, and it is suggested that the symptoms arise from a toxæmia the result of an over-secretion of the thymus gland. And the affection has been called "thymic asthma."

More recently Blumbler ('Trans. Assoc. Amer. Physn.,' 1903, p. 253) has suggested that the toxine is not necessarily the thymus secretion, but arises also from the lymphatic glands—is, in fact, a lympho-toxæmia.

There is still much doubt on the subject, and even if the affection is toxic it is open to question whether the lymphatic-gland enlargement itself is not a result of some toxine possibly absorbed from the alimentary tract.

In my case, the boy being 5 years old was able to struggle against his breathlessness for some three

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days, instead of succumbing at once, as was the case in young infants.

During these three days his symptoms were certainly not due to laryngeal spasm, and the necropsy showed they were not due to any pressure by the thymus gland.

They seemed certainly more like toxic symptoms than anything else, in fact, were not unlike those sometimes seen in what has been called "uræmic asthma."

And I think the case may be deemed worthy of record in this Section, as the history may perhaps be of use in affording a clue to the cause of sudden dyspnœa in cases that may occur in the future.

February 10th, 1908.

FERMENTS AS INTESTINAL ANTISEPTICS.

By ALFRED S. GUBB, M.D.,
Aix-les-Bains and Algiers.

In a more or less vague way yeasts and ferments have been recognised for centuries to possess certain "blood-purifying" properties. In truth the employment of yeast for this purpose belongs rather to domestic medicine and savours of "old women's lore." So-called blood purifiers are, almost without exception, bodies that stimulate digestion or check gastro-intestinal fermentation for the simple reason that by far the commonest source of "impurities" is the alimentary tract, which, under certain circumstances, become a veritable toxine factory, the which when absorbed and excreted excite cutaneous irritation.

Furunculosis, for instance, is practically always of alimentary *qua* digestive origin, and "barm" was, and still is, the popular remedy—and an efficacious one—for it. Abroad, wine ferment—"ferment de raisin"—is empirically employed in precisely similar conditions, and its *modus operandi* is, of course, identical. Still farther afield, half-civilised races, whose food is both copious and indigestible, have come to recognise that great benefit attends the methodical ingestion of fermented milks, the ferments made use of invariably comprising at least one capable of elaborating lactic acid.

Of course lactic acid possesses well-known antiseptic properities, but the anti-putrefactive action of these fermented milks is not due, or only partly so, to the lactic acid they incidentally contain. It is dependent on the introduction into the alimentary canal of robust organisms themselves innocuous to human beings, but which monopolise the soil and oust the so-called "irregular" ferments—butyric acid and the like—which are responsible for the "acidity" of the stomach and for the production of the toxines that give rise to the constitutional symptoms associated with laboured or imperfect digestion.

It may be pointed out just by way of illustration that meat rapidly undergoes decomposition, which proceeds unchecked, whereas milk turns sour, and having done so remains quite a long time free from putrefactive changes. The explanation is that milk is rich in sugar, which undergoes lactic acid fermentation, and this protects it for a time, at any rate, against the germs of putrefaction, whereas meat is not thus protected.

The employment of chemical antiseptics for the purpose of checking gastro-intestinal fermentation, after having had a certain vogue, is now generally discredited because it is recognised to be impracticable to administer them in sufficient quantities to produce the desired effect; moreover, their action, such as it is, is necessarily limited to their sojourn in the canal, which cannot be of long duration.

Fresh yeast is difficult to obtain under ordinary circumstances; moreover, it is of uncertain composition and is very liable to undergo collateral fermentations, which are the despair of the brewer and of the therapeutist alike. It is now prepared in more concentrated and trustworthy forms as *levurine* or *levurine extractive*, the latter representing something like thirty times its weight of the crude product in addition to being easy to take, and, if protected against damp, perfectly stable for an indefinite period.

The lactic acid ferments are also available in agreeable and concentrated forms. Fermented milk is prepared under various names—koumiss, kefir, yahourth, leben, verenatz, etc., and in the form of curdled milk powders, bouillon (cultures), and tablets under the name *lacto-bacilline*.

Apart from their therapeutical properties, which will be referred to later, these products constitute an invaluable addition to the invalid dietary.

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Kefir, for instance, which can be prepared by any skilful chemist, is a pleasant, highly digestible beverage, and it is welcomed by patients as a change to the monotonous insipidity of pure milk. Milk curdled by the aid of lacto-bacilline is also pleasant to take, and as nutritious as it is readily assimilable.

They can be made of varying degrees of lactic acidity, this being dependent on the duration of the fermentation, and the therapeutical effect is influenced by the proportion of acid present therein. Kefir, for instance, is classified as 1, 2, and 3, the No. 1 containing comparatively little lactic acid (twenty-four hours' fermentation) and No. 3 a good deal (three days' fermentation).

Therapeutical applications.—In all cases of gastro-intestinal fermentation, the pathological import whereof has been thoroughly investigated by Huchard, Hayem, and others, these ferments, however employed, afford immediate and durable relief. One of the commonest and most fatal forms of intestinal fermentation is seen in the diarrhœa of young children associated with acute enteritis, diarrhœa, vomiting, anorexia, and fever. Drugs are practically useless, and the constitutional intoxication is so rapid that unless speedily checked another little victim is added to the terrible list of "preventible but unprevented" deaths. In such cases we may make use of curdled milk, prepared according to the formula devised by Professor Metchnikoff, or we may give lacto-bacilline powders mixed with boiled, diluted milk, sweetened with sugar, in order to provide pabulum for the lactic acid germs.

In older subjects flatulence, acidity, heartburn, post-prandial lethargy, and cutaneous lesions dependent on imperfect digestion and irregular fermentation promptly subside under the influence of levurine extractive or lacto-bacilline tablets just as they would if koumiss or kefir were employed.

Flatulent dyspepsia, again, is a fertile cause of insomnia, or, at any rate, of disturbed, unrefreshing sleep. In such cases the ferment acts almost like a narcotic by suppressing the reflexes of intestinal origin that interfere with the slowing down of the cerebral circulation associated with normal sleep.

In obstinate diarrhœa, dysentery, "slippery bowels," and the extensive series of tropical enterites the results yielded by fermented milks, or the ferments which they contain, *e.g.* lacto-bacilline powder or tablets, are, however, even more striking. I have obtained most remarkable effects in Algeria in obstinate cases of quasi-tropical diarrhœa that had resisted all the pharmacopœial remedies that I had brought to bear and which yielded in a couple of days to levurine, kefir, or lacto-bacilline—for I sometimes employ one and sometimes another. Moreover some patients exhibit a certain idiosyncrasy or fastidiousness which renders it desirable to have two or three strings to one's bow.

Surgeon-Major Brochet, who has charge of the Civil Hospital at Haiphong (Indo-China), reports a series of cases of severe enteritis, refractory diarrhœa, and dysentery in patients who were only sent into hospital when outside treatment, in some instances of several weeks' duration, had failed to afford relief. His detailed notes of these cases show how remarkably prompt and efficacious were the effects of a curdled milk diet—the curdled milk being both food and medicine. The particular ferment he utilised was that of lacto-bacilline, which is the most convenient for use in outlying stations by reason of its stability and facility of transport. He adds, too, that condensed milk, which is often the only milk available in hot countries, answers the purpose admirably, the only drawback being that the excess of sugar intensifies the lactic fermentation and may give rise to disagreeable acidity. This, however, can be neutralised by the addition of a little bicarbonate of soda or magnesia. His cases include several of typical dysentery with from twenty to sixty stools mixed with blood in the twenty-four hours, yet these proved just as amenable, though necessarily less rapidly, as the others.

The lactic germs establish themselves in the intestine in the course of a few days—about a week—and can be isolated in the fæces so that, once they have gained a footing, the process of intestinal disinfection goes on, so to speak, automatically.

The use of these ferments has now been placed on a sound scientific basis, thanks to the researches of Huchard, Robin, Hayem, etc., and their utility is such that a very brief trial will suffice to convince those as yet unfamiliar with their dietetic and therapeutical qualities. I would only add that while curdled milk can be prepared by anyone with a minimum of experience and but little trouble the fermented milks such as koumiss, kefir, etc., can only be satisfactorily prepared in a properly equipped laboratory by persons who have acquired the necessary *tour de main*.

February 10th, 1908.

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A CLINICAL LECTURE

ON THE

TREATMENT OF INJURIES OF THE UTERUS.

Delivered at the Middlesex Hospital.

By J. BLAND-SUTTON,
Surgeon to the Hospital.

ON several occasions I have been called upon to deal with severe injuries of the uterus, and as the principles relating to the treatment of these injuries are unformulated, I thought it would be useful to bring the subject under your notice. Information relating to injuries of the non-gravid, as well as the pregnant uterus, is widely scattered in periodical literature, and there is no lack of records, for these injuries are common. They fall easily into four groups:

(1) *Gynæcological injuries.*—This group includes perforations by the uterine sound, dilators, curette, or the nozzle of a syringe, or a douche. In this group we must place cases of accidental introduction of strong antiseptic solutions into the peritoneal cavity.

(2) *Obstetric injuries.*—This comprises such accidents as rupture of the uterus, arising from the use of midwifery instruments, or the operation known as "turning." It will also include the so-called spontaneous rupture of the uterus, due to violent contractions of the organ in obstructed labour.

(3) Injuries to the pregnant uterus, due to falls, kicks from man or other animals; horn-rips of the uterus, bullet-wounds, etc.

(4) Injuries to the gravid uterus in the course of abdominal operations.

The simplest, and certainly the commonest, accident is perforation of the uterus with a sound, dilator, or curette. When the instruments are

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carefully sterilised, and the uterine cavity free from pathogenic micro-organisms, the passage of a sound through its walls is rarely attended with any untoward consequence. A few years ago when the uterine sound passed into the abdomen through the walls of the uterus and no evil followed, it was believed by the credulous that the instrument had traversed the canal of the Fallopian tube. This fable is sometimes related now, but I doubt if any practical gynæcologist seriously believes it. When the sound or the uterus is septic, perforation of the organ has been followed by rapidly fatal peritonitis. The walls of the uterus have been perforated by the sound when used by the most experienced practitioners, so that it becomes necessary to advise those who are learning the principles of gynæcology never to employ this instrument in a routine fashion, and when its use is really necessary take every care to make it aseptic, and render the parts of the patient clean with an antiseptic before passing the instrument. A wound made by a dirty uterine sound may prove as lethal as a snake-bite.

On the whole, injuries by dilators come more frequently under notice because they are more obvious at the time they are inflicted. I have been often consulted by junior practitioners who, after perforating the uterine wall with one of these instruments, has asked my advice as to treatment. When the dilator is small and clean and there is no bleeding the case may be left to itself; in one instance it became necessary to open the abdomen for septic peritonitis; and in another a localised abscess formed in the pelvis, and required drainage through an incision in the posterior vaginal fornix. Occasionally very serious consequences follow simple perforations by dilators and curettes, and this has induced one gynæcologist (G. W. Jarman) to urge that if in the course of dilatation and curettage of the uterus, a rupture or perforation of the uterine wall occurs, it is better to perform a cœliotomy and assure one's self of the safety of the patient than to hope that no untoward result will ensue ('Gynecological Transactions,' Philadelphia, 1905, vol. xxx, 21).

A careful study of the effects of forced dilatation of the unimpregnated uterus shows that if the dilatation is carried beyond No. 8 (Hégar's scale) some tearing of the lateral wall occurs, and often this tearing will extend to and involve the serous membrane. When the operator is clumsy, or inexperienced, or both, it does happen that the serous membrane is torn through without being recognised. This is a serious trap, because it was, and probably is still with some practitioners, the custom after dilating the cervical canal to flush the uterine cavity with an antiseptic solution. When there is a hole in the uterine wall this poisonous stuff will leak into the belly. Some years ago a gynæcologist curetted the uterus of a patient and then flushed it out with a quart of perchloride of mercury solution (1 in 5000). To his astonishment none of the solution returned, and on examining the uterus he found a wide rent in the wall: the antiseptic solution had passed direct into the general peritoneal cavity. The patient died in a few hours. This is not an isolated tragedy, but I am full of hope that it is very uncommon; fortunately it can be avoided if the practitioner, when he curettes a uterus, will be content to mop it out, instead of uselessly deluging the endometrium with antiseptic solution.

Cases are not unknown in which nurses in douching lying-in women have unconsciously perforated the soft wall of the uterus with the hard nozzle of douching apparatus and delivered the poisonous solution direct into the belly.

In many instances in which the uterine wall is torn in the course of instrumental dilatation, the first indication of the accident is the presence of intestines in the rent, and I shall relate to you in the course of this lecture some cases which will prove that *one of the most serious complications of injury to the unimpregnated, as well as to the gravid uterus, is extrusion or prolapse of the intestine.*

Some years ago a practitioner started to dilate and curette a young married woman for dysmenorrhœa; when the cervical canal was sufficiently dilated to admit a finger something soft was felt in the uterine cavity, and the operator proceeded to remove it; thinking the mass was fœtal membranes he diligently pulled on them till both hands were full; one end he cut through and the other he pulled away forcibly. On dropping the supposed membranes into a pail, the doctor found to his horror that the supposed fœtal membranes was small intestine. Eight hours later I was called to the patient, and found her nearly dead from loss of blood. I performed cœliotomy, removed a large quantity of blood from the belly, and secured the

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hole in the uterus with a suture: I then found the cut end of the ileum, and sutured it into the cæcum, for the intestine had been torn away at the ileo-cæcal junction. The abdominal cavity contained neither gas nor fæces. A triangular piece of mesentery measuring nearly two metres in length required excision and sutural union of the cut edges. The abdominal incision was closed with a single row of sutures and drained. The patient recovered.

On another occasion a practitioner, whilst dilating the cervical canal preparatory to curetting, suddenly forced the dilator through the posterior wall of the uterus at the junction of the neck and body of the organ: without recognising what he had done he continued to pass the dilators until he had thrust the biggest instrument (No. 20) through the rent. A fellow practitioner who was assisting him, suspecting something wrong, examined the patient, and found small intestine in the hollow of the Sim's speculum which rested in the vagina. I was sent for, and found a large transverse rent on the posterior aspect of the uterus, and as there was free bleeding it seemed to me wiser to perform cœliotomy: the rent was so extensive as to render subtotal hysterectomy with conservation of the ovaries the safest course.

Extensive injuries of this kind sometimes happen to experienced gynæcologists, and the uterus is exceptionally liable to this injury when dilatation and curetting are performed shortly after miscarriage. One such ruptured the uterus during dilatation, and under the impression that he was removing a soft growth, or secundines, withdrew eight inches of small intestine; he recognised the error and pushed the intestine back through the opening in the uterine wall. The patient recovered and had subsequently had two successful pregnancies.

Professor R. v. Braun-Fernwald has collected a number of remarkable cases illustrating the frequency with which the uterus is perforated by gynæcologists with sounds, curettes, and dilators.

He concludes his interesting paper with this observation: "The dextrous operator may use instruments for which he has a preference; for him the art lies not in the instruments but in the hand."

There is another class of gynæcological operation in which the uterus runs great risk of rupture, namely, in that known as vaginal myomectomy. The removal of an extruded fibroid polypus is one of the simplest surgical operations, but when the uterus of a middle-aged spinster contains a sessile, submucous fibroid, the size of a walnut, it is not always a simple or an easy matter to extract it. Some years ago I published a case which illustrates this very well. A patient had been duly delivered of her second child; coincidently with the expulsion of the placenta the doctor detected a sessile submucous fibroid protruding at the widely-opened mouth of the uterus. It soon disappeared within the cavity as the womb contracted, and during the protracted lying-in gave rise to troublesome profuse bleeding. Seven weeks later I was asked to remove the fibroid. In due course the cervical canal was dilated and I found a fibroid somewhat larger than a golf-ball sessile on the fundus of the uterus. The uterine tissues were soft and easily lacerable. In endeavouring to dilate the canal, the largest dilator tore through the uterine wall at the the base of the tumour. The slit was wide, the uterus soft, friable, and unsatisfactory. The patient was the wife of a doctor, so I explained the situation to him and with his consent performed vaginal hysterectomy. The patient recovered.

Accidents of this kind are by no means rare, and in attempting to perform vaginal myomectomy when the tumour is large and entirely within the uterine cavity, and especially if the patient is a barren woman, the operator is liable to tear through the uterine wall in the process of dilating the cervical canal, or he will tear a hole in the wall whilst dragging the tumour out of its capsule. In some lucky instances no harm results; in the hands of an experienced man untoward effects are averted by a prompt hysterectomy. In the hands of those who trust to luck after such accidents the patient runs a risk of dying from hæmorrhage, or septic peritonitis, and in pre-antiseptic days the mortality after the operation styled "enucleation of uterine polypi" was very high.

Ruptures of the uterus in the process of instrumental dilatation are rarely reported, but there is no gynæcologist to whom this accident has not happened. J. Jakob, of Munich, wrote a thesis in 1905 on the danger of instrumental treatment of intra-uterine disease. He collected 141 instances of perforation of the uterus; of these 23 died, chiefly through septic peritonitis. Among these

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injuries, 73 were inflicted with the curette; 19 with the sound; 16 with dilators; 14 with forceps (Ausraumungszangen); and 6 were due to cathethers (Spülkather).

There was a very instructive discussion reported in the 'American Journal of Obstetrics,' 1906, vol. liv, p. 618, founded on a paper by Dr. Charles Congdon, in which he had collected and analysed a number of reported cases of injuries of the uterus, which show the great liability of the intestine to be injured when the uterine wall is torn. It appears that in one instance the physician, with the object of facilitating labour, attempted to eviscerate the fœtus, but he in reality withdrew the small intestines from the abdomen of the mother. In another case, bowel was mistaken for the umbilical cord and freely pulled out. In most of these severe cases the patients died.

The treatment adopted with slighter degrees of prolapsed bowel, associated with rupture or tears of the uterus, has consisted in washing the extruded bowel or omentum, returning it and trusting to the contraction of the uterus to close the rent. In many instances this has proved successful. On the whole the most hopeful method, and certainly that which has furnished the best results when rupture of the uterus, gravid or non-gravid, has been complicated with prolapse and injury of the bowel, is cœliotomy with suture of the rent, or removal of the uterus if the rent be wide. Resection of the injured bowel may sometimes be required.

Some of the most remarkable injuries inflicted on the gravid uterus are the consequences of attempts to induce what is technically known as criminal abortion, especially when the abortion is self-induced. For example, a midwife, æt. 35 years, mother of four children, but living apart from her husband, was in the third month of pregnancy when she passed a catheter soaked in lysol into the internal os; it slipped, and she lost control of it. The patient died of septic peritonitis. A broken elastic bougie, 13 cm. long, was found in the abdomen under some coils of intestines. Schoenbeck, who reports the case, shrewdly suggests that the bougie was probably introduced by another person.

Some of the most desperate measures are adopted by single women who are pregnant, or imagine themselves pregnant, after illicit intercourse. A young woman about five months pregnant, and in despair in regard to her condition, thrust a knitting-needle into the uterus through the anterior abdominal wall; a surgeon was called in, performed cœliotomy, and luckily found an end of the needle sticking out of the anterior wall of the uterus. He withdrew it, the patient miscarried, but recovered. A patient once confessed to me that she induced abortion on herself by passing a long wooden penholder up the vagina, and when she could not pass it any further with her fingers sat down on the holder and forced it in. Abortion and a severe illness followed, but she recovered.

In studying a large number of records relating to injuries of the uterus, it is remarkable to find that in some instances a simple perforation will destroy a woman's life in two or three days. In other instances, unpleasant or dangerous consequences are long-delayed; for example, Petrquin and Foltze reported a case in which a midwife introduced a sound into a pregnant uterus; abortion followed. Four months later a swelling formed at the navel; this was incised, and a uterine sound extracted.

The most extraordinary assault upon a pregnant uterus for the purpose of inducing abortion I have ever read of was perpetrated by a widow, æt. 29 years; when in the fifth month of pregnancy she fired a revolver-bullet into the uterus through the anterior abdominal wall. Cœliotomy was performed by Hans Kehr, and the wound in the uterus closed by suture. The patient aborted fourteen days after the operation, but recovered.

A gravid uterus in the later months of pregnancy is a big organ, and like the abdominal viscera generally may be severely damaged by blows, kicks from horses or brutal men, butts from animals, such as a calf, or a goat, falls upon the belly, or a fall downstairs, or the woman may be run over. The treatment to be adopted in these conditions varies widely with the circumstances. As a general rule it may be stated that the most satisfactory mode of treatment is cœliotomy; this permits a thorough examination of the organ, and facilitates removal of effused blood. In the late stages of pregnancy accidents of this kind entail Cæsarean section.

Among the most curious injuries of this group are those known as horn-rips: these are cases in which the pregnant uterus is torn open by the horn of a bull. An interesting collection of cases illus-

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AN INDEX OF TREATMENT

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trating this accident, under the title of 'Cattle-horn Laceration of the Abdomen and Uterus of Pregnant Women,' is furnished by Robert P. Harris. Even after very severe injuries, in some of which the intestines protruded, women have recovered, and several of the children survived this terrible mode of delivery.

There is another consequence of injury to the gravid uterus which needs mention, and it may be illustrated by an actual case recorded with great care by Leopold:

A woman when near the mid-period of pregnancy injured herself by slipping down the cellar steps. When she arrived at what she reckoned to be "term," extra-uterine pregnancy was diagnosed and cœliotomy performed. A fœtus of about the fourth month of gestation, enclosed in a thin amniotic sac, was found in the abdomen, and its umbilical cord passed through a rent in the back of the uterus. The uterus and placenta were removed with success. In this instance the uterus was ruptured by the fall, and the fœtus in its membranes slowly entruded into the peritoneal cavity. Cases of this kind in connection with uterine pregnancy are very rare in women. Several similar examples have been carefully observed and recorded in connection with tubal pregnancy.

In mammals, such as the cat, rabbit, sheep, and dog, many instances have been described in which mummified fœtuses in their membranes have been found hanging from the omentum, or lying loose in the peritoneal cavity. These "abdominal fœtuses" are due to rupture of a gravid uterus, and the fœtuses in their membranes are slowly extruded through the rent in the uterine wall into the peritoneal cavity, where they become quietly encysted, and are found after death by the veterinary surgeon, an anatomist, or a butcher. These cases were supposed to be examples of primary extra-uterine gestation, but many years ago I gave the explanation described above, and it has been amply confirmed by Pembrey, Bellingham Smith, and Kamann.

Rupture of the uterus during labour.—The uterus is liable, during labour, to be torn as a result of its own expulsive efforts, especially when the transit of the fœtus is hindered or obstructed by narrowness of the pelvic outlet, tumours, or undue size of the child. This form of injury is called *spontaneous rupture*, to distinguish it from rupture due to instruments, such as midwifery-forceps, cephalotribes, etc. The uterus is not unfrequently torn in the obstetric manœuvre known as "turning."

It is very important for the practitioner to recognise rupture of the uterus. For example, Jenkins reports a case in which a practitioner performed version without an anæsthetic; he then introduced his hand into the uterus to remove the placenta and his fingers were touching the liver before he realised what happened. There was nothing in the patient's condition to indicate such a serious complication. Hysterectomy was performed, but the patient died three days later. The literature relating to this accident is abundant, and the reports issued from lying-in institutions deal with extensive figures, but unfortunately the reporters are not in harmony on the principles of treatment.

Ivanoff has made a careful study of the results which follow the various modes of treating rupture of the uterus during labour in the Moscow Maternity Hospital, from 1877 to 1903. During this period there were 124 examples of this accident. He comes to the conclusion that surgical intervention gives the best results, as it permits hæmostasis and cleaning of the wound ('Annales de Gyn. et d'Obstet.,' 1904).

The gravity of this accident is well shown in a paper by Munro Kerr, containing an analysis of fourteen cases classified according to the method of treatment:

Nature of the operation.	No. of Patients.	Maternal Deaths.
Plugging (all incomplete ruptures) . . .	3	0
Hysterectomy (with extra-peritoneal treatment of the stump)	1	1
Subtotal of hysterectomy .	5	3
Total hysterectomy . .	3	2

Two patients died without any operation being undertaken ('Brit. Med. Journ.,' 1907, vol. xi, p. 445.)

A. von Walla collected from the Frauenklinik at Buda Pesth 28 cases of rupture of the uterus—seventeen incomplete and eleven complete. The incomplete cases were treated conservatively and ten died. Of the eleven complete cases six treated conservatively died; five were operated on and two recovered. Von Walla thinks that complete rupture of the uterus always indicates operation ("Ruptura Uteri Completa, Abdominale totai

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Extirpation," Heilung, 'Centralblatt für Gynäkologie,' 1900, p. 497.

There are three methods of dealing with rupture of the uterus:

(1) Treat the patient conservatively, which means at most lightly packing the part with antiseptic gauze.

(2) Performing cœliotomy and stitching up the rent in the uterus.

(3) Hysterectomy, preferably by the abdominal route, as this enables the peritoneal cavity to be cleared of clot.

The only point in which there is any semblance of agreement among obstetricians is this: In cases of complete rupture in which the fœtus and membranes are extruded from the uterus into the belly cœliotomy is clearly indicated.

Klien (Dresden) published in 1902 a critical study based upon 367 cases of rupture of the uterus published in the preceding twenty years. Of these cases 149 were operated upon with a mortality of 44 per cent.; 198 were not operated upon, 96 recovered and 102 died—a mortality of 52 per cent. Among the unoperated cases some were not treated in any way, and in these the mortality was 73 per cent., whilst those treated by drainage, plugging and irrigation, the mortality was only 37·5 per cent.

When there is dangerous bleeding Klien advises immediate operation. Lacerations of the vagina make the prognosis unfavourable, and especially injury of the bladder.

Klein's paper ('Arch. f. Gyn.,' 1901, Bd. 62, p. 193) is the most complete analysis of statistics dealing with any kind of accident that I have ever read. During the last ten years hysterectomy has been so much improved and the technique simplified, that the operative treatment of complete rupture of the gravid uterus will be more frequently undertaken in the future than it has in the past, and with every prospect of reducing the heavy bill of mortality at present associated with this grave accident.

Injury to a Gravid Uterus in the course of an Abdominal Operation.

In spite of every care it has happened on many occasions that a pregnant uterus has been mistaken for an ovarian cyst, the abdomen has been opened and a trocar plunged into the uterus. In some instances a uterus in which the pregnancy has advanced as far as the sixth month has been removed under the impression that it was a large ovarian cyst, and this accident has happened with a pregnant uterus greatly enlarged in the somewhat rare condition known as hydramnios. A pregnant uterus is also liable to be stabbed by an ovariotomy trocar when the condition is complicated with unilateral or bilateral ovarian cysts. The gravid uterus has very thin walls and, occasionally, resembles so very closely an ovarian cyst as to deceive an inexperienced operator.

When the surgeon finds that he has injured a pregnant uterus in the course of an abdominal operation three courses are open to him, each of which has been practised with success by surgeons of renown:

(1) Sew up the incision in the uterus.

(2) Perform Cæsarean section.

(3) Remove the uterus (subtotal hysterectomy).

Several cases have been reported in which injury to a gravid uterus during ovariotomy has terminated fatally, especially when the surgeons followed the plan of sewing up the wound in the uterus.

A careful consideration of the reported cases indicates that the best results follow for the patient when the surgeon performs Cæsarean section, as the following record shows:

Sir Spencer Wells had removed a large, multilocular ovarian cyst from the left side of the patient, when he felt what was supposed to be a cyst of the right ovary. When tapped it was found to be a gravid uterus in which pregnancy had advanced to near the fifth month. Cæsarean section was at once performed and the patient recovered.

Injuries of this kind are rarely likely to happen now, for the clumsy ovariotomy trocar is passing out of use.

Those who are interested in this matter will find a series of abstracts of cases published in my work 'On Surgical Diseases of the Ovaries, etc.,' second edition, p. 396.

Bullet-wounds of the Pregnant Uterus.

These are very rare, and, like rupture of the uterus, liable to be complicated with injury of the intestines; it is for this reason that the canon of surgery applicable to penetrating wounds of the

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abdomen should be practised in these circumstances, and the patient submitted to cœliotomy.

When the gravid uterus is penetrated by a bullet there may be little bleeding on account of the contracting property of the uterine tissue. In some instances amniotic fluid stained with blood escapes. In operating, the anterior as well as the posterior surface of the uterus should be carefully examined in order to determine if the bullet passed through

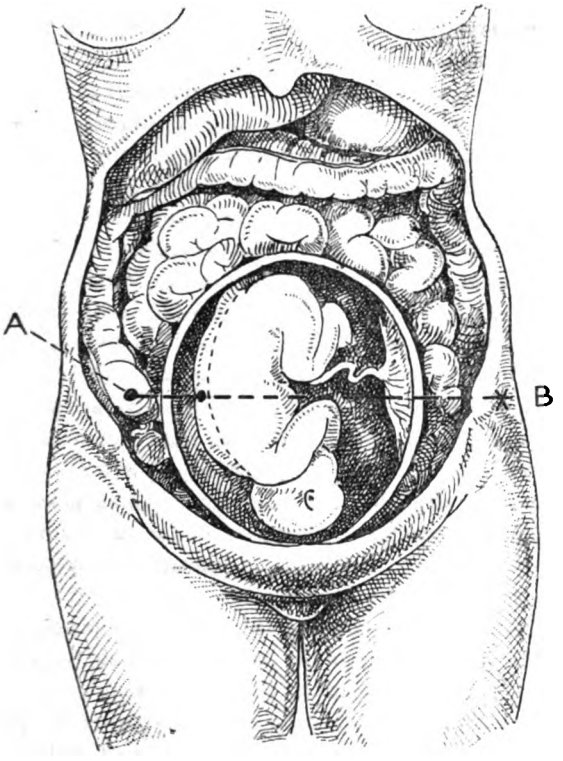

FIG. 1.—A diagram to represent the course of the bullet in Prichard's patient (from a drawing by T. Carwardine). She was æt. 28 years, and in the seventh month of pregnancy. The bullet was extracted from under the skin on the left side, four inches behind the anterior superior spine of the ilium. (From the 'British Medical Journal,' 1891, vol. i, p. 332.)

this organ. In some instances the fœtus has been injured by the bullet. When free bleeding follows a bullet-wound of the gravid uterus the hæmorrhage is usually due to damage of blood-vessels connected with the intestines.

The best method of dealing with the uterus in such conditions is undetermined. In a case recorded by Prichard a revolver-bullet traversed the uterus, the fœtus, made six holes in the small intestine, and wounded an artery in the mesentery. Hysterectomy was performed, and the holes in the intestine closed with a purse-string suture. The patient died on the sixth day after the operation from peritonitis (Fig. 1).

In a case reported by Albarran, the patient was æt. 19 years and in the fifth month of pregnancy when shot. There were four perforations of the small intestines, and the mesenteric artery was wounded. He resected 20 cm. of small intestine. A loop of umbilical cord protruded through the bullet-hole in the uterus; this was resected and the ends of the cord tied. The patient miscarried a few hours after the operation, but recovered.

Baudet reported a case in which there were four perforations of the small intestine. This surgeon sutured the wounds in the uterus and the holes in the bowel; the woman aborted some hours after the operation, but recovered.

In a case under Robinson's care the bullet entered the uterus and penetrated the right shoulder of the fœtus. The patient, who was in the eighth month of pregnancy, quickly miscarried. The bullet was found in the *débris*. The patient not only recovered, but reconceived, and gave birth to another child in the following year.

February 17th, 1908.

THE PNEUMOCOCCUS INFECTIONS.*

By WILLIAM OSLER, M.D., F.R.S.,
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THE pneumococcus infections alone among the maladies of modern life have increased in frequency and severity. The mortality from diphtheria has fallen in twenty-five years more than 75 per cent.; the death-rate from typhoid fever has fallen still more, and that of tuberculosis is steadily declining. Making due allowance for unavoidable errors in the figures, there can be no question of the enormous increase in the death-rate from the most important of the pneumococcus infections, *i.e.* pneumonia, particularly in the United States. In some cities the mortality has increased fourfold. In this country, too, the death-rate has steadily increased

* Delivered before the Medical Society of London.

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during the last decennium, but not in the same startling ratio as in the large cities of the United States. In Chicago, for example, the mortality from pneumonia has reached as high as 20 per 10,000 of inhabitants, one eighth of all the deaths being due to it—46 per cent. more than from all other contagious diseases combined. As the Registrar-General's report shows, it is in the cities that the great increase has taken place, and there are evidently conditions in our modern life favouring the spread of the infection. What these are is not easy to say, but they are conditions unassociated with drainage and water supply. While segregation of population has increased, the concentration has lessened, and household sanitation has everywhere improved. It is not easy to settle upon any one outside factor responsible for this remarkable change. It is a question worthy of the most careful study—all the more so because of its obscurity. In cities the element of frequent contact comes into play, in the school, in the street, in the tramcars, etc., and in modern life this may be an important factor in the promotion of the spread of this special type of infection. Certain diseases, the plague for example, have great waves of incidence, associated possibly with an increased virulence of the germ, and we may be upon the crest of a pneumococcic wave of such length that we lack the date for its measurement.

We are here face to face with a most serious situation in preventive medicine, one quite as deserving of organised study as cancer or tuberculosis. The work of the past ten years has put us in a position to attack the problems with renewed vigour. There are still many lacunæ in our knowledge of the life-history of the pneumococcus. As is the case with so many germs, the more carefully they are studied the greater are found to be the variations, and we have learnt to recognise varieties in the species pneumococcus, which differ not only in their morphological characters but in degrees of virulence. The relation of these forms to one another and the relation of the whole group to the streptococci is being studied in many places. The work of the New York Pneumonia Commission has done much to clear up the relation of the typical and atypical forms, but it is a question of great difficulty which may take years for its solution.

One of the most interesting of the many transformations which bacteriology has made in clinical medicine has been the extension of our knowledge of the diseases caused by the pneumococcus. Inflammation of the lungs is only one, though the chief, among a score or more of important and serious diseases due to it. Unless it be the genococcus, no organism has risen so rapidly in the scale, and it may be called the David of bacteria, killing its tens of thousands to the thousands of any other.

Pneumococcic septicæmia is met with (1) in pneumonia. With the new methods of blood culture we have found that in a considerable proportion of all cases the blood swarms with the organisms. The toxæmia and many of the complications may be associated with this bacteriæmia, which, curiously enough, does not always disappear with the crisis; secondly, though rarely, as a fatal septicæmia without recognisable local disease; thirdly, in the endocarditis and other local pneumococcic lesions; and lastly, as a terminal infection in various chronic maladies. One of the first cases of this sort to come under my notice was a child with an acute tuberculous process at one apex. A sudden exacerbation of the fever with general œdema and coma gave a picture somewhat suggestive of a tuberculous meningitis, but the autopsy showed an intense pneumococcal septicæmia.

Naturally affections of the respiratory passages take the first place in the group. As I shall state later, a majority of us carry about a potential infection. Catarrhal and suppurative affections of the upper air-passages are not infrequently caused by pneumococci. There has recently been described a remarkable epidemic of ophthalmia with catarrhal symptoms, caused by a closely allied organism, and the great importance of infection of the accessory sinuses has been dwelt upon by StClair Thomson and others. The meningitis arises, in some cases at least, from this source. A bronchitis of singular chronicity may be associated with an infection of the tubes following a pneumonia. A girl, æt. 14 years, had pneumonia at the age of ten, a very severe and obstinate attack. For four years she had cough of a peculiarly irritating character, two or three spells in the day, in which she brought up a small quantity of viscid sputum. The history, of course, suggested a bronchiectasis. The physical examination was negative, save for the presence of

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a few piping râles at the bases of the lungs. The sputum in smears was as if taken from pure culture of most typical pneumococci.

Next to the tubercle bacillus the pneumococcus plays the most important part in inflammations of the pleura. Acute fibrinous pleurisy and empyema are the chief forms, both the result of direct infection from the lung, either from a frank pneumonia or quite as often from small undiscoverable patches. The great increase in the frequency of empyema of late years has been noticed by some observers. In some of the large Metropolitan Hospitals, for example at Guy's, as described by Hale White, the cases have doubled in number in ten years. This has been attributed to the influence of influenza, but it has been just as marked in regions in which this disease has not been very prevalent. The special features of the pneumococcal empyema cannot here be discussed, but of all serious infections by this group of organisms, it is one with the lowest mortality when taken in hand early and properly treated.

The great respiratory infection, the most formidable acute disease of modern times, well called, in Bunyan's phrase, the "Captain of the Men of Death," is inflammation of the lungs, from the association with which the pneumococcus has had its most popular name. A few points only may be indicated for consideration: the causes of the undoubted increase of the disease; does a septicæmia precede the development of the local lesion as the investigations of Rosenow would suggest? the question of an increase in case mortality, the question of frequency and variety of clinically atypical forms; and lastly, how far are we in this generation treating the disease better than our fathers? I say our fathers, as I have no doubt about the lethal character of the treatment of our great grandfathers.

Of cardiac affections due to the pneumococcus, the endocarditis, by far the most serious, is either associated with pneumonia or is met with as an independent disease; at least, we cannot always find a primary source. With anatomical features of its own, it is perhaps more often dextral than the endocardial lesion of any other organism, and in a high degree it shares with the streptococcus forms the property of malignancy. Very few cases recover, and very many are overlooked clinically, as the cardiac features of the case may be completely masked in a profound toxæmia.

Peritonitis is the most fatal of the abdominal infections of this group. Much more common than statistics indicate, it has several peculiarities—a high mortality, a high incidence in childhood, a relative great frequency among female children, a tendency to localisation and a stormy clinical course resembling that of the acute perforative peritonitis.

Pneumococcic meningitis, with many features of interest, comes third in order of frequency among the acute inflammations of the cerebro-spinal meninges. So far as I know, it is one of the few infections of the group, besides pneumonia, that occurs in epidemic form, and in very much the same way, in small house outbreaks of from three to five cases. As a complication of pneumonia it is not uncommon, occurring sometimes with the endocarditis. Isolated instances are met with in which no other lesion is found, and it is quite possible that the infection, as in the epidemic cerebro-spinal fever, may come through the nose or the accessory sinuses. In contrast to this latter form, it seems to be invariably fatal, at least that has been my experience.

Besides these chief affections of the respiratory, circulatory, gastro-intestinal, and cerebro-spinal systems, there is a large group of minor maladies due to the pneumococcus—abscesses, subcutaneous, muscular, periosteal, arthritic and certain cases of otitis media; some of these are of small account, others are the source of widespread and even fatal infection.

Source of infection. — It has been known for many years that pneumococci exist in the mouth and throat of a certain number of healthy people. Indeed, Sternburg made his original observations on the production of mouse septicæmia by the injection of human saliva. The recent studies by the New York Pneumonia Commission have added greatly to our knowledge of this subject. Park and Williams examined 200 cases, chiefly normal individuals, and found pneumococci present in a large proportion of all, whether resident in the city or in the country. In fifty out of fifty-three cases of lobar pneumonia cases examined the organisms were present, and in the majority, of the typical virulent variety. It is interesting to know that a larger number of atypical strains were obtained from healthy persons than from patients with pneumonia. Longcope and Fox, of the same

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Commission, had 83 per cent. of positive results in forty-two individuals. One of the most interesting points in their observations was the increase of the percentage of typical pneumococci in the mouth secretions during the months of December and January. In several instances the study of the saliva of the same individual between the months of November and April showed a remarkable change. The organism obtained in December was very virulent and killed mice, while in April the organisms were much less virulent, so that they would produce no effect whatever. On the other hand, they found that some persons always had virulent pneumococci in their mouths. Buerger, at Mount Sinai Hospital, New York, reported to the same committee a study of seventy-eight persons and the percentage of positive results was about fifty. The pneumococcus may appear suddenly in a normal mouth. The period of persistence was variable, but in some persons it was found with great regularity. Buerger made a study of the communicability of the organism from one person to another—so far as I know, the first attempt of the kind that has been made. The evidence was sought by demonstrating the absence of pneumococcus in the mouths of certain cases, and by studying the possible sources of subsequent infection. For this study two male wards, one containing twenty-four and the other twelve beds, and a medical children's ward were selected. The number of positive cases present in the small ward at any given time was rapidly ascertained. It was repeatedly found that normal individuals—that is, those in whose mouths the pneumococcus was repeatedly found to be absent, acquired the organism by association with cases of pneumonia or with positive, normal persons. To take an example, the patient in Bed I was examined on December 4th and 10th and found negative. On that day to the next bed a patient with lobar pneumonia was admitted, in whose mouth virulent organisms were repeatedly demonstrated. Five days later and on a number of days subsequent the pneumococci were detected in the mouth of the adjoining patient.

Buerger brings forward evidence to show that handkerchiefs and "positive normal" cases may be regarded as means of transportation of the pneumococcus from one person to another. A point of some moment brought out in a paper by him in the same series was the appearance and existence in two cases of pneumococci in the mouths, coincident with the development and course of ordinary colds.

It has long been known that the viability of pneumococcus was not very great, though it depends largely upon how the sputum is treated. In a study made of the whole question for the New York Commission, Wood found that in moist sputum kept in a dark room the average life of the pneumococcus was eleven days, but if exposed to direct sunlight, and if it dries and is desiccated, they rapidly die. He found that persons suffering from pneumococcus infections, in coughing, sneezing, expectorating, or talking, expelled from the mouth particles of sputum or saliva which contained pneumococci and which might remain suspended in the air for a number of hours if the ventilation of a room was not good. They become harmless in a very short time, one and a half hours being the limit.

The upshot of this, and of other work, is to show the exceedingly wide prevalence of pneumococcus, and that the great majority of healthy individuals, particularly in the winter months, harbour pneumococci in the secretion of their mouths and throats. The way by which the pneumococcus reaches the lungs has not yet been fully determined. The common explanation is that when the resistance of an individual is weakened from any cause, the pneumococci present or inhaled reach the lungs and excite inflammation. In view of the recent researches on the modes of transmission of tubercle bacilli by Grober and others, the infection may pass through the lymphatics, and we know also that the plague bacillus reaches the lung through the lymphatics of the neck and gives rise to a pneumonia. On the other hand, the work of Rosenow suggests the possibility of a hæmatogenous infection, as there may be a bacteriæmia before a local lesion in the lungs is detected. Altogether there is much work to be done yet on the paths of infection in pneumonia.

The two important facts which have been brought out are the widespread prevalence of the pneumococci in the mouths of apparently healthy individuals, and the extraordinary variability which the germs show in virulence. We know, too, that there are great variations in resistance of the different races of men

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to the pneumococci. The negro in my wards at the John Hopkins Hospital showed a death-rate from the disease nearly 50 per cent. above the white, and throughout the Southern States of America this somewhat remarkable susceptibility is manifest. A clinical experiment on a very large scale has been going on for several years in South Africa. Dr. Porter's study of the disease of the Chinese and Kaffirs in the Rand mines shows that, while the former have a susceptibility to pneumonia quite equal to that of the negroes in America, the Chinese show a resistance far above that of the whites, and there have been in South Africa epidemics of severe pneumococcus infection characterised by catarrh of the upper air-passages and gastro-intestinal disturbances, while pneumonic lesions were rare.

Wadsworth, in a series of most interesting experiments, produced for the first time, I believe, pneumonia in rabbits subjected to a certain degree of immunisation. Then we know that individuals at different ages present different degrees of susceptibility. The newborn and the infant are resistant, the adult and the middle-aged are susceptible, and debilitating influences, as exposure, alcoholic habits, render persons susceptible. It may be a battle between the degree of resistance and the grade of virulence of the organism harboured. There are facts which indicate that the atypical strains produce lung lesions which differ in degree, at any rate, from the true lobar pneumonia. A score or more of questions of the most intense interest and of the greatest practical importance are raised by these recent studies upon this most formidable of all infections. One point of the greatest practical importance remains, viz., Is it possible to remove the sources of danger from the secretions of so many healthy individuals? A complete disinfection of the mouth is impossible. A very careful study of the effect of different solutions on the vitality of the pneumococci in the mouth has been made by Wadsworth, who found that of all mouth disinfectants that which has alcohol as a basis is the most effective. He recommends an alcoholic solution of bicarbonate of soda and sodium chloride with the addition of glycerine, made as strong as the patient can stand. It is to be remembered that the pneumococci not only live in the saliva and the secretions of the mouth, but in the throat and in the crypts of the tonsils—regions in which it is still more difficult to reach.

February 17th, 1908.

ALCOHOL IN THE TREATMENT OF PNEUMONIA.

By G. A. SUTHERLAND, M.D., F.R.C.P.,
Physician to the North-West London Hospital, and to Paddington Green Children's Hospital.

In considering the question of the value of alcohol in this disease one is met at the outset by two difficulties, first, that there is no agreement as to the cause of the cardiac failure, which is the reason for giving alcohol, and secondly, that there is no agreement as to the action of alcohol on the circulatory and nervous systems. For instance, it has been held by different writers that the cardiac failure is due to some form of inflammation of the heart, to obstruction of the pulmonary circulation, to poisoning of the medullary centre, and to the general toxæmia. Now, if alcohol in any form and in any amount is a poison to the system, as some maintain, and if the patient is already suffering from a toxæmia which is depressing the heart, it is plain that the combination of two poisons will not prove beneficial unless one is the direct antidote of the other.

It may be urged that plenty of time and opportunity has now been afforded to settle the question as to the use of alcohol in pneumonia, but the question is not so simple as it appears. It is not a case of treating pneumonia, it is a case of treating a patient suffering from pneumonia, and patients vary in their susceptibility to alcohol just as they do to other drugs. One class of patients may respond well to alcohol as a stimulant, in another it may act as a depressant, in a third it may provoke gastro-intestinal disturbance, and so on. Each observer will tend to be influenced in his judgment by the results which occur most frequently under his own observation, and as the experiences of various observers may differ very considerably, their conclusions may appear to be diametrically opposed. Again, the cardiac weakness may be due in one case to pre-existing heart disease, in another to pneumococcal carditis, in another to pulmonary obstruction, and so on. These are complications, and not the ordinary conditions of pneumonia which we are now discussing. Regarding the use of alcohol in the treatment of pneumonia we are not justified in laying

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down any dogmatic rules which will apply in all cases. We must, however, have certain views as to the nature of the cardiac failure, and as to the action of alcohol, if our treatment is to be anything but empirical. For the sake of clearness two assumptions will be made in the following remarks: first, that the commonest cause of cardiac failure in pneumonia is poisoning and paralysis of the medullary centre, and secondly, that alcohol is in the majority of cases a stimulant of that centre. These are assumptions, and time will not allow of my going fully into the reasons for adopting them, but a large amount of clinical evidence may be adduced in support of them.

What is the proper time for giving alcohol? At the beginning of an ordinary attack of pneumonia in a previously healthy or moderately healthy subject there is no occasion to give alcohol. Some commence the routine treatment of pneumonia with three or four ounces of brandy a day, and assert that cardiac debility must be averted by early treatment with alcohol. A special characteristic of alcohol is that it acts quickly, so that one has not to wait a few days for its effects to be perceptible, as in the case of digitalis. In the early days of a pneumonic attack alcohol should be withheld, and the patient's strength should be sustained by suitable food. After the fourth day or when the signs of the pre-critical stage present themselves, alcohol may, with advantage, be given and continued through the exhausting period of the crisis, until recuperation has set in. The period during which alcohol may be beneficially given will thus be limited in ordinary cases to from three to six days. Its action is strictly comparable to that of the other stimulants which may be used at the same time. After the crisis has passed the amount of alcohol may, as a rule, be rapidly diminished.

The first indication for the use of alcohol is given by the pulse. When the pulse becomes small or irregular or compressible and runs up to 110 or more per minute alcohol is called for. The more suddenly this change takes place the more urgent is the call for alcohol, as the whole system is injuriously affected by a sudden impairment of the circulation. Confirmatory evidence will be found on examining the heart, when the first sound at the apex may be found to be weakened or blurred, and some dilatation of the left ventricle is present. Unless some evidence of cardiac weakness is recognised it is not advisable to give alcohol. Other associated disturbances of the system may indicate a call for stimulation, for instance, sleeplessness. This can often be relieved by a full dose of alcohol. Again, the cardiac weakness may induce a disturbed state of the secretions, as shown by a dry mouth and tongue. Here also alcohol may afford relief. There may be low muttering delirium, with restlessness, owing to the weakened circulation through the brain, a condition which is often benefited by alcohol.

Having decided to use alcohol for one or other of the above indications, one must give it a fair trial and watch the result closely. The stimulant is not to be continued on any general principles, but on the indications which the progress of the patient affords as to whether it is acting beneficially or not. If the pulse and heart improve, if the secretions become re-established, and if the delirium or restlessness or sleeplessness is removed, then the alcohol is probably doing good and is to be continued as required. If, on the other hand, one fails to perceive any improvement under the use of alcohol in sufficient amount, it is better to discontinue it and trust to other forms of stimulation. It should be kept in mind that many cases of pneumonia can be successfully treated without alcohol at all, and that the indications for its use should be as clear as in the case of any other powerful drug.

There are certain contra-indications which must be referred to. In the presence of a full and bounding pulse it is folly to give alcohol as a cardiac stimulant, or for any symptom such as restlessness, or sleeplessness, or delirium, or loss of appetite. If there is reason to suppose that pneumococcal myocarditis, or endocarditis, or pericarditis, is the cause of the cardiac weakness, alcohol will not be called for in large doses, and it is questionable whether under these conditions stimulation by alcohol will benefit the heart. My own experience has been that in acute inflammation of the heart the less alcohol one gives the bette . If there is considerable blockage of the pulmonary circulation and embarrassment of the right side of the heart, these conditions should be relieved by other measures—cupping, leeching etc.—before alcohol is used. The place of alcohol is to strengthen the action of the left ventricle after the

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state of the pulmonary circulation has been relieved. If there is much pulmonary œdema or bronchial catarrh the free use of alcohol will rather tend to aggravate the condition, unless it is traceable to weak action of the left ventricle. If the patient is prostrated by profound toxæmia any attempt to revive him by large doses of alcohol will probably prove useless. His blood and tissues, already saturated with the poison of pneumonia, will neither be purified by means of alcohol nor rendered more capable of throwing off the toxines. As already stated it is the collapse produced by failure of the cardiac centre, and not that caused by general toxic infection, which will be benefited by alcohol. If the effect of alcohol is to excite and disturb the patient, to increase the distress, or to fail in producing an improvement in the cardiac embarrassment, it is advisable to reduce the amount or to discontinue it altogether.

The form in which the alcohol is supplied is important. Some have advised the use of the stronger wines, port and sherry, because of the special ethers they contain, and of the added nutritive effect as compared with a plain spirit such as brandy. The ethers can hardly be determined beforehand by the doctor, and the chief point to be considered is the stimulating effect of alcohol. In the case of wines the strength in alcohol varies so much that one cannot regulate accurately the amount of alcohol which is to be given. A sound brandy or whisky is to be preferred as allowing of exact alcoholic dosage, and as supplying the stimulant in a form which is suited to most stomachs. If the patient or his doctor has a preference for rum or gin, equally good results may be obtained by either of these. On the grounds of palatability and quickness of effect champagne has also distinct claims, and it may sometimes act better than brandy in disturbed states of the stomach. After the crisis has passed and the stage of convalescence has been entered on, port or sherry or Burgundy may be employed in place of the spirit.

The question of dosage is one to be determined by the special requirements of the case. Speaking generally, one may say that a mild amount of stimulation will be secured by three ounces of brandy or whisky in the day, a moderate amount by six ounces, and a full amount by nine ounces. If it be said that these would be large amounts for a person in perfect health it may be replied that the conditions in pneumonia are quite different. It is well known that many patients suffering from pneumonia can take large quantities of alcohol without the production of those symptoms which would certainly follow from the same amounts taken in a condition of good health. The object of the stimulation is to raise the force of the circulation as nearly as possible to that existing in health, and the dosage is to be regulated on this principle. Again, it may be said that the effect of large doses of alcohol on the tissues generally has been found to be injurious. To this it may be replied that such an effect depends on the length of time during which the absorption of alcohol has been going on. The question of alcohol in pneumonia differs from that in most other affections in that it is a self-limited disease, and in that the period of stimulation may be put down as from three to six days. It is a case of treatment during a critical period, and we are justified in pushing our remedies in a way which would not be possible or advisable in a case of prolonged illness. From this point of view the above-mentioned amounts are not excessive, and if the stimulating effect is beneficial no permanent harm can be done to the system by the alcohol. These amounts will not, in the vast majority of cases, require to be exceeded. At the same time, if the physician is convinced that the alcohol is benefiting the patient, and that more is called for, he should have the courage of his convictions and increase the quantity.

It is advisable to give the brandy or whisky in doses of from two to four drachms, at regular intervals, and diluted with at least twice the amount of water. The aim is to produce a steady and continuous stimulative effect. Larger doses tend to upset the digestion, and to produce a temporary over-stimulation followed by a reaction. Sometimes, however, a condition such as sleeplessness or delirium may be best treated by one full dose of from one to two ounces.

February 17th, 1908.

We have received from Messrs. Ilford some of their "Ilford X-ray Plates" and find that the makers' claims concerning their excellence are borne out by actual experience. In the essential requirements of shortness of exposure and clearness of detail these plates answer every test, and they are further characterised by that most desirable quality in plates, viz. uniformity.

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FRACTURE OF THE ACETABULUM.

By R. W. FELL, M.B., C.M.Edin.

As fractures of the acetabulum are so rare I think it may prove interesting to many to have the report of a case which came under my notice a short time ago. As to the diagnosis the accompanying radiograph will speak for itself.

The facts are these: A lady, æt. 31 years, was proceeding along the road in a high dogcart, the coachman driving, when the horse shied and swerved to the right, running the wheel up a steep bank on the side of the road. The dogcart overturned, bringing the lady with violence to the ground on her left hip. The coachman fell on to her right hip. Thus the left great trochanter was crushed on to the ground by a combined weight of at least twenty stones plus the force of gravity acting through about five feet. At the moment of the fall she felt practically no pain, but on attempting to rise she felt her leg to be quite helpless and her hip extremely painful. A passing wagonette conveyed her home, a distance of six miles, and I saw her on arrival.

Except for a moderate abrasion on the left temple, all injury was centred in the left hip, which was kept flexed, being the position of greatest ease. Any attempt at extension, active or passive, caused extreme pain. There was also pain, evidently reflected, extending down towards the ischium and to the groin. Under chloroform the flexion of the hip disappeared, but not until the anæsthesia was fairly deep and all reflex spasm abolished. No shortening of the limb was appreciable, and abduction and rotation were quite absent. The marked feature of the case was flattening over the left great trochanter, with preservation of its contour. It seemed to have receded inwards. Measurements of the distances between the anterior superior iliac spine and great trochanter on the two sides showed marked inequality. On gently rotating the thigh crepitus of a soft nature was distinctly appreciable The soft parts around the hip showed no signs of bruising externally, but deep-seated effusion was easily felt. It was clearly indicated that serious damage had been done to the hip-joint, and it was obviously a case for consultation. I accordingly asked Mr. Arbuthnot Lane to see the patient with me. Radiographs were taken, and the interesting fact was proved that the head of the femur had

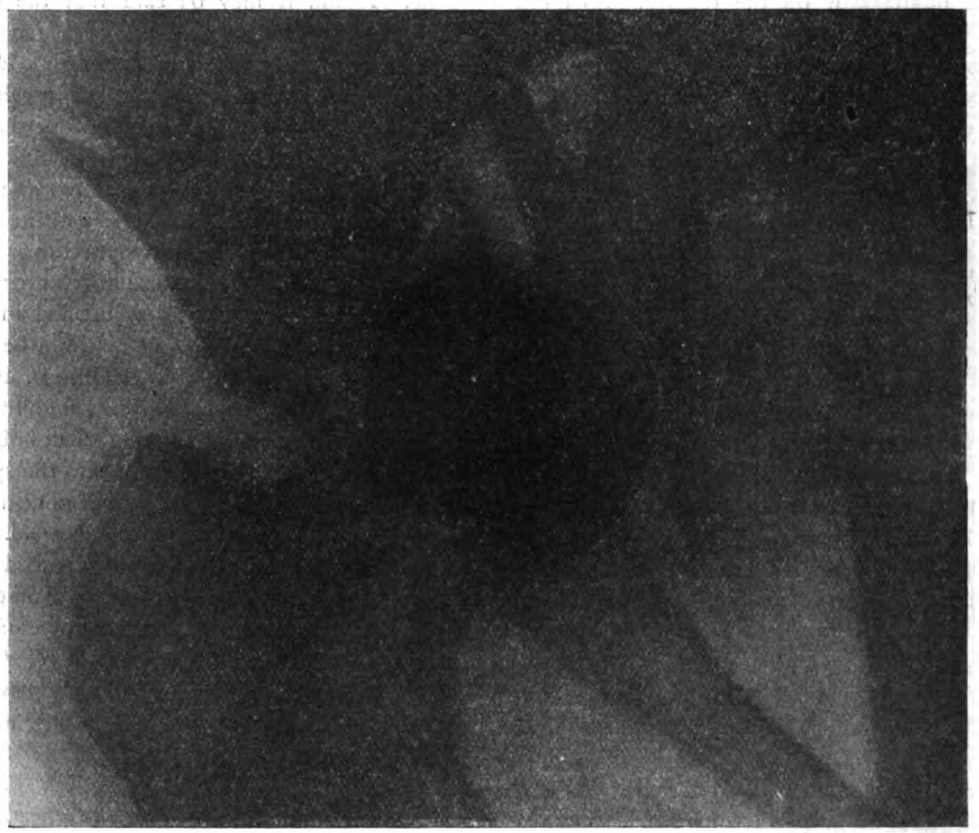

FIG. 1.—Before treatment.

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passed right through the acetabulum and was lying actually in the pelvis. Mr. Lane then proceeded to reduce the displacement, which he effected by manipulation from within the pelvis *per rectum* and vagina, combined with leverage on the femur, and traction. He distinctly felt the head of the femur in the pelvis along with comminuted fragments of bone. After due replacement, extension was applied through the medium of plaster, and weight, and pulley, and the foot of the bed raised. Radiographs were again taken, which showed the head of the femur in its normal position. This case is interesting from the point of view of the "mechanics" of fractures. The primary factors must be direction of force, amount of force and strength of material. In this case the patient must have alighted completely on her great trochanter, which transmitted the full force of weight and gravity through the neck and head of the femur, right on to the thin lower and front part of the acetabulum. This is in all subjects the weakest part, and in some subjects it is extremely thin, particularly the whole of the acetabular fossa. Above and behind, however, the acetabulum is thick, strong and wedge-like, an obvious provision for the concentration upon it of the body weight.

The radiography in this case was unusually difficult on account of the large effusion of blood, which is shown in the radiographs here reproduced by a dark shadow for some distance around the joint. My thanks are due to Mr. Coldwell for his radiographs, which proved of great practical value.

The subsequent course of this case was highly satisfactory. The extension weight of six pounds was retained for four weeks and caused no discomfort. It was then removed and the limb subjected to massage. The patient was then allowed to get up and was soon able to take gentle exercise on crutches. At the end of twelve weeks she was able to move about comfortably with a stick. Flexion at the hip-joint was now free, and although not completely restored was steadily increasing. A comparison of the contour of each hip showed perfect symmetry, and the relations of the great trochanter were perfectly normal. All that was now required was continued judicious exercise and movement to restore the normal muscular tone, and to loosen any adhesions around the joint that still remained.

FIG. 2.—After treatment.

February 17th, 1908.

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WITH MR. ARBUTHNOT LANE AT GUY'S HOSPITAL.

THE following interesting case of an ununited fracture of the left tibia was operated on by Mr. Arbuthnot Lane at Guy's Hospital. The patient was a man, æt. 23 years, who fractured his tibia in a fall from a horse; the injury was treated with splints and plaster for seven months, but the man was unable to walk. On admission to Guy's Hospital a fracture could be felt about three inches below the tuberosity of the tibia, the fragments of bone being freely movable on one another. The line of the fracture appeared to be oblique, extending from above outwards and downwards. There were two reddish and tender spots, one on the inner and the other on the outer aspect of the seat of the fracture, and bony prominences could be felt beneath them. Mr. Arbuthnot Lane made an incision on the anterior aspect of the leg immediately over the fracture, exposing the ends of the bone. These ends were freed from tissue and about one inch of bone was removed, part being taken from each of the ends. The sawn surfaces of the bone were then brought into apposition. Mr. Arbuthnot Lane then made another incision over the outer side of the fibula, which was exposed and sawn through. The two ends of the tibia were then pushed together and a Y-shaped metal plate was fastened by means of screws on to the anterior surface of the bone; a dumb-bell-shaped metal plate was also secured by screws to the outer side of the tibia. Both wounds were then closed with Michel's clips. The results of the operative treatment of this case were in every way satisfactory.

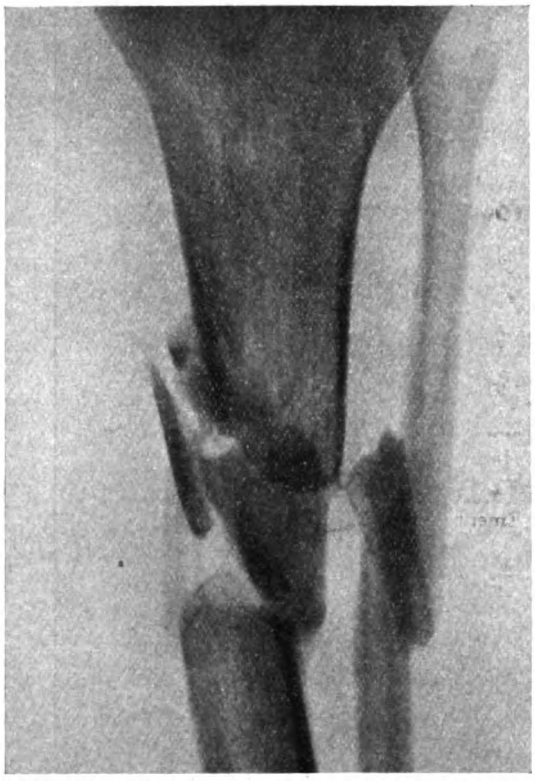

FIG. 1.—Before operation.

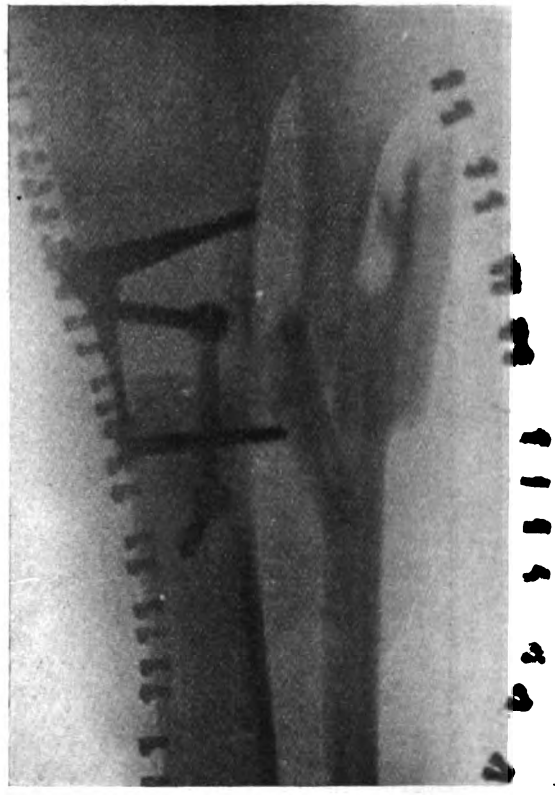

FIG. 2.—After operation.

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A CLINICAL LECTURE

ON

UTERINE FIBROIDS COMPLICATING PREGNANCY.

Delivered at University College Hospital.

By G. F. BLACKER, M.D., F.R.C.P.,

Obstetric Physician to University College Hospital.

GENTLEMEN,—I have chosen the subject of fibroid tumours occurring with pregnancy, because it is not an uncommon condition, and though it is true that in the majority of such cases there is no interference with pregnancy or delivery, yet this complication at times may be a source of serious danger to the mother and to the child. With regard to the frequency of the condition in my last 200 cases in private practice I have seen 5 cases of fibroids complicating pregnancy. In the Clinique Baudelocque in Paris, Pinard found 85 cases of fibroid tumours causing definite symptoms in 13,814 cases of pregnancy, or a frequency of ·62 per cent. If a fibroid tumour complicates pregnancy it may do so by influencing the expulsion of the fœtus, or by interfering with the development of the fœtus while in the uterus, or by causing dangers in the puerperium. Of the 85 cases recorded by Pinard, 66 of the patients went to full term, 13 were delivered prematurely, and 5 aborted. In 54 of the cases delivery took place spontaneously, only 24 out of the 85 cases required instrumental interference, and in 65 of the cases the children were born alive. These figures prove that even when pregnancy is complicated by large fibroid tumours, in the majority of the cases delivery occurs spontaneously without any trouble.

We will consider the subject in the following order: The relation of these tumours to sterility, the influence of the pregnancy on the tumour, the influence of the tumour on the pregnancy and the

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labour, the complications occurring during the puerperium, and finally the diagnosis and treatment of the condition.

With regard to the relation between fibroid tumours and sterility, it used to be said, and it is still maintained by some writers, that fibroids are a cause of sterility, and statistics have been brought forward to prove this contention. But generally it can be shown that in sterile women who have fibroids the tumours have given rise to symptoms or begun to grow at a comparatively late age, and the sterility in reality ante-dates the occurrence of the fibroid. Of course you are aware that sterility in a woman is most important between the ages of twenty and thirty; but the majority of women do not develop large fibroid tumours, until they are between thirty and forty years of age. So it is difficult to say that fibroid tumours are certainly a cause of sterility. It is much more likely that the sterility is the cause of the fibroid tumour, rather than the reverse. The view, then, that sterility is caused by fibroid tumours may be given up in favour of the opposite view, namely, that it is sterile women who develop fibroids. It is probable, however, that a fibroid tumour which may have been present for a long period of time before causing any symptoms may play some part in the production of absolute sterility, or of "one-child" sterility, that is to say, the class of case in which a woman has one child and no more.

With regard to the variety of fibroid tumour which is likely to favour sterility, small subserous tumours can have but little influence. Large interstitial tumours and tumours growing in the cervix may well interfere with the occurrence of pregnancy. That submucous tumours may produce sterility is not surprising when you remember that such tumours are usually accompanied by changes in the mucous membrane of the uterus, leading to the occurrence of excessive hæmorrhage and leucorrhœa—both important factors in the production of partial or complete sterility. Subserous tumours are not infrequently accompanied by pelvic peritonitis and inflammation of the appendages, which may lead to sterility. So you will see it is at least likely that fibroid tumours tend to cause sterility, although it is probable that the sterility in women with fibroid tumours probably ante-dates the fibroid in most cases.

When a woman with a fibroid tumour becomes pregnant, what are the changes which are likely to take place in the fibroid? In most cases there will be some change in the position of the tumour, more particularly in the case of those growing from the anterior wall of the uterus, because it is easier for the fibroid to be drawn up above the short anterior pelvic wall than above the sacral promontory. Extreme softening of the tumour is the rule during pregnancy. The fibroid may become so soft that it can no longer be recognised as a tumour, and it is possible for this change to become so marked in advanced pregnancy that the pressure of the child's head flattens the tumour out to such an extent that it no longer forms any obstacle to delivery. A good example of extreme softening of the tumour during pregnancy is afforded by the case of a patient of mine, 30 years of age, who has a fibroid of the uterus of the size of a six months' pregnancy. There was very marked softening of the tumour present in the later months of the pregnancy, and during the labour it contracted very markedly with each uterine pain. The contractions could be readily seen and felt. The history of this case is of interest. Soon after her marriage the patient consulted an obstetric physician who recommended her strongly to have hysterectomy performed because of the dangers which he said would ensue if she became pregnant. She, however, did not act on that advice, preferring to run the risk of pregnancy as she was very anxious to have children. As a matter of fact she has had three children, and in each instance the labour has been quite free from any complication. Even now she will not have the tumour removed, because she wishes to have another baby, one of her children having died, although, as might be expected, the presence of so large a tumour has given rise to very marked separation of the recti muscles and laxity of the abdominal wall.

If the tumour is of the subserous variety, especially if it is pedunculated, twisting of the pedicle may occur as a result of pregnancy. The following case, lately in Ward 3, illustrates this point. The patient was four months' pregnant and was admitted into the hospital with the symptoms of an abdominal tumour with a twisted pedicle. There was a history of a sudden attack of acute abdominal pain with vomiting, and besides the

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pregnant uterus a tender lump was found in the abdomen which was thought to be a dermoid cyst of the ovary with a twisted pedicle. I show you the specimen, which was removed by abdominal myomectomy; it proved to be a subserous pedunculated fibroid attached to the uterus by a pedicle one and a half inches in width. The patient made a good recovery, was delivered spontaneously at full term, and is now in good health. Another effect of the occurrence of pregnancy with a fibroid tumour may be the production of very severe pains in the tumour. An interesting example of this is afforded by the history of a patient who was under my care last year at the Great Northern Hospital. The woman was three months' pregnant, and had a number of small subserous and interstitial fibroids in the uterus. She complained of very severe pain in the abdomen, so severe that it prevented her from sleeping; she gave a history of loss of flesh, and there were occasional slight rises of temperature for which we found no cause. I thought it very probable that she had some peritonitis in connection with the subserous fibroids. Under treatment in the hospital she improved very markedly, the pain became less, she slept better, and her weight increased. In these circumstances I recommended that she should be allowed to go to full term, and that then, if necessary, an abdominal section should be performed. She went to full term, but did not return to the hospital for her confinement. During labour there was uterine inertia, and the application of forceps having failed the medical attendant performed internal version and effected the delivery of a stillborn child with considerable difficulty, the mother dying two hours afterwards of shock. It is very important to remember that version in the case of fibroid tumours complicating pregnancy is, as a general rule, contra-indicated. It is possible that if this patient had been delivered by Cæsarean section the result might have been more favourable. At the same time we must remember that her condition throughout was a very serious one, and she was at no time well enough to stand a major operation of any kind without great risk. In the cases where such severe pain is present it is due either to tension in the capsule caused by the rapid growth of the tumour, or to accompanying attacks of pelvic peritonitis.

The changes produced in a fibroid tumour by the occurrence of pregnancy may be summed up as follows: Alteration in the position of the tumour, softening of the tumour, twisting of the pedicle of a pedunculated subserous tumour, and severe attacks of pain due to the rapid growth of the tumour or to accompanying attacks of pelvic peritonitis.

Next, we must consider the influence of the fibroid tumour on the pregnancy. It happens occasionally that a woman with fibroid tumours has an extra-uterine pregnancy, and I have here two specimens of this kind from the museum. An assertion which is commonly made is that fibroid tumours in a pregnant uterus tend to increase the liability of the patient to premature delivery or abortion. This is a statement of very doubtful accuracy. Indeed it is more likely that fibroid tumours are not a cause of abortion, as in thirty-six cases recorded by Hofmeier thirty-two of the patients went to full term, and only four, or 12 per cent., aborted. The frequency of the occurrence of abortion in ordinary cases of normal pregnancy is difficult to estimate; it can only be determined from accurate observations of large numbers of cases in private practice, because the cases which come into hospital are those in which the abortion produces symptoms, and large numbers of women have an abortion without being aware of the fact. But it is usually estimated, and probably correctly, that one abortion occurs to every five full-term confinements, in other words in 15 to 20 per cent. of all cases of pregnancy, so that the presence of a fibroid tumour hardly can be said to be a powerful factor in the production of abortion if it occurs in only 12 per cent. of these cases. The importance of this conclusion you will see when we consider the treatment of this condition. Another complication which may occur is an abnormal position of the placenta. It is not uncommon to find the placenta attached to the fibroid tumour or to the lower uterine segment. Placenta prævia occurs in about 1 in 500 cases of normal pregnancy; but it occurs in 1 in 120 of the cases which are complicated by fibroids. That justifies us in saying that a fibroid tumour in the uterus may be a cause of placenta prævia. The possibility of the attachment of the placenta to a fibroid tumour is an important complication, because it may lead to severe post-partum hæmorrhage. If the tumour is a submucous one the mucous membrane over it tends to atrophy, and

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the attachment of the placenta to such an area of atrophied mucous membrane may result in the chorionic villi becoming intimately united to the muscular tissue of the uterine wall. Cases have been recorded in which it has been found impossible to remove such an adherent placenta, and where hysterectomy has had to be performed, and the whole uterus removed with the placenta *in situ*.

As we might anticipate mal-presentations are common when fibroids of the uterus complicate pregnancy. The frequency of vertex presentations of living and dead children at full term is about 95 per cent., of breech presentations 3 per cent., and of transverse presentations ·5 per cent., while with fibroid tumours the frequency of these presentations is respectively about 54 per cent., 24 per cent., and 19 per cent.; so that breech and transverse presentations are very much more common in these cases than in normal pregnancies.

The influence of a fibroid tumour upon the development of the fœtus is but a slight one. You might imagine that a large submucous or interstitial tumour would tend to produce marked deformity of the fœtus, but as a matter of fact, no doubt in part as a result of the extreme softening which takes place in the tumour, any such deformity is a rare occurrence. In a case with a number of interstitial fibroids I saw last year, the patient, who presented no symptoms, was delivered naturally at full term of a fœtus with marked talipes calcaneus, which disappeared with massage and manipulation. You must bear in mind that a fibroid tumour, though coexistent, may not be responsible for the occurrence of such a deformity.

Abnormal hæmorrhage from the uterus is the next complication we must consider. This is not uncommon, and naturally suggests a threatened abortion. In the majority of cases, however, the hæmorrhage ceases, and as we have seen these patients are not especially prone to abort. Another complication is an attack of pelvic peritonitis; this is especially likely with pedunculated subserous tumours. Further, during pregnancy the tumour may give rise, as we have already said, to excessive pain. This is the result either of the influence of the pregnancy on the tumour, or of the tumour on the pregnancy. But, at any rate, the fact remains that in these cases there is often severe abdominal pain during the pregnancy. Three explanations of this may be given: The pain may be caused by intermittent uterine contractions, or by tension on the connective tissue of the capsule, owing to the rapid growth of the tumour, or by attacks of pelvic peritonitis around the tumour. If a patient has a very large tumour complicating pregnancy she may get pressure symptoms. I show you a uterus containing a number of fibroid tumours, which were removed by total abdominal hysterectomy by Dr. Herbert Spencer, to whom I am indebted for the opportunity of showing you the specimen, from a patient who was eight and a half months pregnant. She had marked pressure symptoms, the abdomen was greatly distended, the limbs and abdominal walls were œdematous. There was a quantity of free ascitic fluid present, and the urine contained albumen. A further effect these tumours may have on the pregnancy is to predispose the patient to incarceration of the gravid retroverted uterus. Last year a patient was admitted into Ward 3 with an incarcerated gravid uterus, containing a small fibroid tumour, 2½ in. in the lower diameter, in the lower uterine segment. The uterus was pushed up with some little difficulty, the pregnancy went on to full term, and the patient was delivered in the hospital without any further complication.

Now, what influence is a fibroid tumour likely to have during labour? In considering this question it is necessary to consider separately fibroid tumours of the upper uterine segment and those of the lower uterine segment. As a general rule the former give rise to no complications during labour at all. If any abnormal condition is present it will be uterine inertia, or possibly post-partum hæmorrhage, especially if the placenta is attached to the tumour. If situated in the lower uterine segment the tumour may produce rigidity of the cervix, or, most important of all, may obstruct delivery. Happily, such a serious complication is uncommon. Of the eighty-five cases which I mentioned as recorded by Pinard fifty-four were delivered spontaneously, and in only twenty-four cases was any instrumental interference required. These figures remind us that a fibroid tumour, whether it lie in the upper or in the lower uterine segment, in the majority of cases gives rise to no difficulty at all during delivery, and if situated in the lower segment of the uterus, either rises up above the pelvic brim in the early stages of labour or is already above the brim before labour comes on. The difficulties which might arise from

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the position of the tumour in the pelvic cavity or below the presenting part of the child are usually overcome in one of three ways. In the majority of cases during the later months of pregnancy the fibroid tumour is drawn up out of the pelvis. We have already seen that one of the most important effects of pregnancy on fibroid tumours is such an alteration in their position. And this is a most important fact to remember in connection with the treatment of these cases. Generally, if it is a submucous tumour or a tumour of the cervix which is obstructing labour, it is not uncommon to find that the tumour is pushed down in front of the presenting part and extruded during delivery. In some instances it is actually born before the fœtus, and in others it is pushed down and then removed by the medical attendant. The third way in which the obstruction caused by a fibroid tumour during delivery may be overcome is by the softening which takes place during pregnancy. Even if the tumour is still in the pelvic cavity, it may become so soft, œdematous, and easily compressible that it offers no longer any obstruction to the passage of the child. Therefore, even if there is a fibroid tumour in the pelvis or in the lower uterine segment, it need not necessarily cause any obstruction to delivery since it may be drawn up out of the pelvis, or it may be pushed down and born before the child itself, or removed, or become so softened that it no longer offers any obstruction to the passage of the child through the pelvic canal.

If the fibroid tumour causes difficulty in the delivery and operative interference is necessary there are certain dangers which may result. There is the increased danger of septic infection, a danger of post-partum hæmorrhage from the uterus, the remote danger of rupture of the uterus, and, as in the case I have already mentioned, the risk of death from shock after delivery.

In some of the text-books the dangers of fibroid tumours complicating pregnancy have been greatly exaggerated. Many gynæcologists teach that a fibroid tumour complicating pregnancy is a very fatal condition, but, as a matter of fact, the evidence obtained from the carefully recorded statistics of lying-in hospitals shows that it is neither very fatal nor very dangerous. The results obtained by Pinard, which I have quoted to you, prove that the presence of fibroid tumours does not, as a rule, give rise to any complication of labour or of the puerperium. It is true that in past years the maternal mortality was a high one—nearly 50 per cent., but it is now, even in the worst cases, not more than 10 or 12 per cent., and in favourable conditions the risk is much less than this.

Now we may consider the diagnosis of a fibroid tumour complicating pregnancy. This may be very simple, or it may be difficult. If there are a number of small sebserous tumours they are mistaken readily for fœtal parts, especially if the tumours have become considerably softened during pregnancy. If the fibroid tumour is a small one the diagnosis, as a rule, is simple, whereas if there is a large tumour present, with early pregnancy, the diagnosis may be very difficult. You must rely upon a careful inquiry into the history, especially as regards amenorrhœa, the rapid growth of the tumour, and a painstaking examination of the abdomen. A history of rapid increase in size of the tumour, of morning vomiting, and of amenorrhœa is very important in the diagnosis of the condition. With regard to the physical signs, mammary changes, softening of the cervix, with purple coloration of the cervix and vagina, will be of great value. A safe clinical rule is that in every case of rapid growth in the size of a fibroid tumour in a patient who might be pregnant you should suspect the possibility of such an occurrence.

The following case is a good example of a tumour of rare occurrence simulating a fibroid tumour of the uterus. The patient was pregnant, and had a solid, fixed tumour of bony hardness in Douglas's pouch. She had been told some years previously at another hospital that she had a fibroid tumour of the uterus, and taking this history into account the swelling in Douglas's pouch was diagnosed as a calcified fibroid. She was allowed to go to full term, and as the tumour did not permit of the delivery of the child through the pelvis I performed Porro's operation. The child was born alive, and is now a healthy boy, æt. 11 years, and the mother made a good recovery. A week after the operation the wound just above the stump of the uterus gave way and a number of hydatid cysts were discharged, the tumour being in reality a calcified hydatid. Such a tumour is a very uncommon condition as compared with a calcified fibroid tumour, and as the patient had been

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informed some years previously that she had a fibroid tumour in Douglas's pouch the error in diagnosis was excusable.

A correct diagnosis often is not made, and of 191 cases recorded by Nauss in only 15 had the diagnosis been made before pregnancy. In 47 the diagnosis was made during pregnancy, and in 61 during labour. In 42 it was only made after labour, and in 26 it was not made until a post-mortem examination was carried out. It is evident, therefore, that difficulties in diagnosis are not uncommon, and it is not an infrequent event for a fibroid tumour complicating pregnancy to escape recognition. In the following case the tumour was not diagnosed until after the labour. The patient, who had had two children, was delivered of her third child after a normal labour. The uterus remained unduly large and the medical attendant thought that possibly a dead twin fœtus had been retained *in utero*. I saw the patient on the third day after her confinement and found the uterus hard and considerably enlarged, evidently containing a large interstitial or submucous fibroid tumour. As there were no symptoms no treatment was required. Three or four months later she was admitted into the hospital with the symptoms of a sloughing submucous fibroid. This was readily enucleated and she made a good recovery. The doctor had diagnosed a twin pregnancy in this case, and that is a not uncommon error which you must bear in mind. As a general rule the diagnosis is fairly easy, and difficulties only arise when there is a large multinodular fibroid complicated by early pregnancy. Many cases have been recorded in which hysterectomy has been performed, and after the removal of the uterus it has been found to contain a fœtus. You must rely, as I have said, chiefly on the change in the colour of the cervix and vagina, the presence of amenorrhœa, morning vomiting, and changes in the breasts. In the later months the other signs of pregnancy can usually be recognised and the diagnosis is not, as a rule, difficult.

The next important question we must discuss is, What is the correct treatment of these cases? Obviously a pregnancy complicated by the presence of fibroid tumours may present almost any variety of complication, and such a case may require at the time of delivery any one of the obstetrical operations. As a general rule the case should be left alone until the patient arrives at full term. It rarely happens that a woman who has fibroid tumours and at the same time is pregnant requires any operative interference until labour occurs. I regret to say that at the present day a very common line of treatment is to perform hysterectomy for fibroid tumours complicated by early pregnancy. In my opinion this is wrong, first because it is impossible to predict in the early months of pregnancy what is likely to happen at full term, and secondly because such a mode of treatment takes no account of the life of the child. Except in cases where urgent symptoms are present threatening the life of the mother you have no right to sacrifice the life of the unborn child for fear of possible complications which may occur at full term. You must consider the life of the fœtus as well as the life of the mother. As far as possible you must allow the pregnancy to go to full term, or at any rate until such time as the child is viable, and then carry out such treatment as will save not only the life of the mother but also that of the child.

If the patient has no symptoms during her pregnancy you should leave her alone. If she has symptoms, adopt whatever treatment may be right to relieve the symptoms. But to remove an early pregnant uterus containing fibroids because of possible complications which may occur when full term has been reached is unjustifiable, especially when you remember the series of cases recorded by Pinard which I have already mentioned, in which 54 out of 85 women were delivered spontaneously, and in only 24 was any operative interference necessary. These figures prove—and I could multiply them from the statistics of other lying-in hospitals—that the great majority of fibroid tumours complicating pregnancy do not require treatment during pregnancy or labour, and therefore the patient should be allowed to go to full term to be confined spontaneously if possible.

Are there any conditions calling for interference during pregnancy? Is it ever necessary to terminate the pregnancy or to remove the uterus with the fœtus *in utero*? There are certain indications for interference during pregnancy, but they are few and occur but rarely. If the patient suffers from attacks of peritonitis, if the tumours are growing very rapidly, and causing interference with the functions of the neighbouring viscera, or if the case is complicated by cardiac or renal symptoms,

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with extreme dyspnœa, severe pain, or other marked indications of pressure, interference may be justifiable. Now, if you do decide it is necessary to interfere, shall you do so by inducing abortion or premature labour, or is it best to perform abdominal section and remove the uterus with the fœtus *in situ*? One thing which you should not do is to induce abortion or premature delivery, as the results obtained by either of these procedures are exceedingly bad. Of 307 cases reported by Lefour, in six labour was induced, and of these cases one mother and five of the children died. In fifty-seven cases recorded by Phillips the pregnancy was artificially terminated three times and two of the mothers died; so that these nine cases of interference were attended with a maternal mortality of 33 per cent.—a mortality which indicates the very grave danger attending the induction of abortion or of premature labour in such cases.

If it is necessary, then, to interfere during the course of the pregnancy—and, as I have said, I consider that this is only justifiable if the life of the mother is actually threatened—abdominal section should be performed and total or sub-total hysterectomy carried out as the case may require. In every instance, however, where you can possibly do so, wait until the child is viable, and then to preserve the life of the child do a Cæsarean section followed by total or sub-total hysterectomy. Let your aim be not only to save the life of the mother but to save the life of the unborn child as well. In the majority of cases of pregnancy complicated by fibroids the woman can be allowed safely to go to full term, and then you can decide with certainty what is the best treatment to adopt. The great importance of such delay is that fibroid tumours situated in the lower uterine segment, as full term approaches, are often lifted up above the brim of the pelvis and no longer form any obstruction to delivery, or if they do not rise up they may become so soft that they are flattened out by the pressure of the presenting part, or if submucous in character they may be extruded in front of the presenting part of the child. Therefore it is important to wait until full term has arrived before deciding what is to be done.

The principles which should guide you in your treatment are, first that you should remove the tumour when you can, secondly, if you cannot remove it you should attempt to push it up above the pelvic brim, and thirdly, if both these modes of treatment are impossible, then you must carefully determine what amount of obstruction to the delivery of the child is present and deal with the case accordingly. How can you remove the tumours? Those which are easiest to remove are fibroid polypi presenting in the vagina. They can be readily dealt with by the vaginal route, and if possible this should be done before labour comes on. If there is a subserous tumour or an interstitial tumour which requires removal you must remove it by abdominal myomectomy or by enucleation. As a rule these operations are carried out during the pregnancy. But unless there are very definite indications for interference it is better not to perform any such operations during pregnancy. An example of myomectomy performed during pregnancy is the case I have already mentioned to you, in which there were symptoms of a twisted pedicle and where the diagnosis was not made correctly. If it is not possible or advisable to remove the tumour, then an attempt must be made to push it up out of the way if it is likely to obstruct delivery. Attempts at reposition are best made just before full term or in the early stages of labour. If you wait until near full term or until labour has commenced you may find that reposition of the tumour is not necessary, because it is very likely to rise up spontaneously. But if it happens that the tumour has neither gone up spontaneously nor can be pushed up or be removed, you have to consider how much room is left for the passage of the child and whether it can be born by the natural passages or not. As a general rule, if there is obstruction to the delivery of the child from the presence of fibroid tumours in the cavity of the pelvis, any attempt to deliver by the natural passages is likely to be attended by greater danger to the mother than delivery by means of abdominal section. The results obtained after a difficult delivery by forceps or version are very bad. In 39 cases in which forceps were used out of 388 cases of fibroids complicating pregnancy, 33 per cent. of the mothers died, and for this reason a difficult forceps delivery in fibroid tumours in the lower uterine segment is contra-indicated as a general rule. Nor is the performance of version good treatment in these cases, as the mortality after version is higher even than that after the application of forceps. Of 81 cases in which version was performed for

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fibroid tumours complicating pregnancy, 53 of the mothers died. It is obvious, therefore, that in cases of fibroid tumour in the pelvis obstructing labour delivery by abdominal section is the safest course. You can perform Cæsarean section alone or Cæsarean section followed by complete or partial removal of the uterus, or Porro's operation. Cæsarean section without removal of the uterus is generally contra-indicated, because it is better not only to deliver the patient, but to free her from the risk of any further pregnancy.

We may therefore recapitulate the treatment as follows: as a general rule do not interfere during pregnancy, except when there are definite indications present, such as very rapid growth of the tumour, peritonitis, or marked pressure symptoms. If you have to interfere during pregnancy, perform abdominal section and do not induce abortion or premature labour; either removing the uterus with the fœtus *in situ* or removing the tumour alone if possible. In the majority of cases you can, however, allow the patient to go to full term. When this period has been reached labour in a large number of the cases will occur spontaneously. If it does not, then, if the tumour is in the vagina and can be removed, remove it; if not, or if it is in the pelvic cavity obstructing the birth of the child, attempt to push it up out of the way. But you must take care to remember that there is danger associated with this method of reposition, and if you use excessive force you may tear the peritoneum or the vessels in the capsule of the fibroid or you may tear through peritoneal adhesions. If you fail to push the tumour up, ascertain as accurately as possible what room is left for the delivery of the child, and if there is any likelihood of delivery by the natural passages being very difficult, perform abdominal section. In coming to a conclusion remember the high mortality attending forceps delivery or delivery by version. In these cases if you decide to perform abdominal section you have a choice of four operations: simple Cæsarean section, Cæsarean section followed by total or sub-total hysterectomy or Porro's operation. The decision as to which of these operations you shall perform must depend on the indications in each particular case. As a general rule, the operation of election at the present day is delivery of the child by Cæsarean section followed by total or sub-total hysterectomy. But there are certain cases in which it is better to carry out Porro's operation: for example, if you have not good assistants, if the patient is not in the best possible surroundings, or if there is any question of her being infected already, Porro's operation is the safest to perform.

The mortality of these operations in cases of fibroid tumours complicating delivery is not more than about 8 per cent., and such a mortality contrasts very favourably with that occurring after the induction of premature labour, version, or the use of forceps. Supposing, however, that the patient has been delivered by the vagina (spontaneously or with operative interference), what are the possible dangers after delivery? The most important danger is retention of the placenta or an adherent placenta, with or without post-partum hæmorrhage. Of 21 recorded cases (in which the placenta was adherent), 13 of the mothers died. The following case illustrates the dangers of this accident very well. The patient had a small subserous fibroid and was delivered at full term by a drunken midwife, who left an adherent placenta *in utero* for some hours before sending for a doctor. Severe post-partum hæmorrhage occurred and the placenta had to be removed manually. Septic infection followed and an attack of puerperal insanity. After many months of acute illness the patient finally recovered. For her second and third confinements she was admitted into the hospital, and was delivered on each occasion of a living child without any complications.

Lastly, we must briefly consider the changes that may occur in fibroids during the puerperium. One of the most interesting and favourable of these is a marked diminution in size which at times accompanies the involution of the uterus. In other cases the lochial discharge may be excessive or there may be secondary post-partum hæmorrhage. Sloughing of the tumour followed by its spontaneous expulsion may occur or may require its immediate removal. Submucous or interstitial fibroids may become infected during delivery and lead to the development of septic infection or septic intoxication. One of the conditions in which hysterectomy is justifiable in puerperal septic infection is when the patient has a sloughing interstitial or submucous fibroid of the uterus, which cannot be removed without the uterus. Occasionally after delivery suppuration arises in a fibroid tumour.

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A specimen of this nature I show you. The patient was under my care at the Great Northern Hospital. After her confinement she developed suppurative anterior pelvic cellulitis; the abscess was opened above the pubes and she made apparently a perfect recovery. The uterus was noted at the time to be enlarged and was thought to contain a fibroid tumour. A few months afterwards she returned to the hospital complaining of pain and a swelling in the abdomen. Total abdominal hysterectomy was performed, and on cutting the tumour open it was found that there was a large cavity containing a slough and about half a pint of pus in its interior. It is, I think, extremely probable that the infection of the tumour took place at the time of the attack of pelvic cellulitis.

In conclusion, I wish to impress upon you more especially that the dangers of fibroid tumours complicating pregnancy have been considerably exaggerated, and that in the majority of cases labour takes place spontaneously without any complication. In dealing with these cases you should, whenever possible, allow the pregnancy to proceed to full term; you must consider the life of the child as well as that of the mother, and in cases where undue difficulty is likely to be met with in any attempt to deliver the child through the natural passages, Cæsarean section is likely to give the best results.

And again, I wish to repeat what I have said already, that the removal of the uterus with the fœtus *in situ* is only justifiable during the early months of pregnancy when the conditions are such as to directly imperil the life of the mother, since in these circumstances only are you justified in destroying the life of the fœtus. The fear that possible complications may occur later on is no justification for such a procedure.

February 24th, 1908.

Cure of Nervous Eructations.—A patient of Saito for thirty-six years had been accustomed to have an hour or so of loud and annoying eructations every evening. If she did not thus get rid of the "gas" in her stomach she was unable to sleep. He convinced her that the trouble was due to her habit of swallowing air, and cured her by making her wear a gag to keep her mouth open, with a vessel to catch the saliva, during the hours when she previously had had the attacks.—*Journ. A.M.A.*, vol. l, No. 5.

THE INDICATIONS FOR SURGICAL INTERFERENCE IN ACUTE ABDOMINAL DISEASES.

Delivered before the Oldham Medical Society.

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MR. PRESIDENT AND GENTLEMEN,—When I set out to put my ideas on this subject upon paper I soon found that it was going to be a very difficult matter, for although I can at the bedside as a rule come to a decision as to the right course to adopt in any particular case, it is quite another matter to satisfactorily reduce to general terms the mental process by which that decision is arrived at.

In fact, after much careful thought I have come to the conclusion that it is impossible. For let us for a moment consider what we have to do when we are confronted with one of these acute abdominal cases, and have to answer the question—should an operation be performed? We have to take into consideration a large number of factors—points in the history of the case, symptoms, and physical signs. Each of these factors in the first place tells either for or against the advisability of operation. But the answer to the question is not to be found by *simply* balancing the factors numerically. For we must remember that all the factors in a given case vary as regards their relative importance, so that the presence of certain factors which are for operation should outweigh certain other factors against operation; for instance, the presence of muscular rigidity may be more an indication for operation than a pulse of only 80 is an argument against it.

But we have to go further still, for the factors in the case not only vary in relative importance as regards one another, but their relative importance varies in different cases. For instance, in the majority of these cases a pulse-rate of 120 is an indication for operation, yet all will admit that there are cases when such a pulse-rate may be entirely neglected. The crux of the whole question as it appears to me is the ability to rightly assign to each factor its correct value in each individual case, and it is this that I find myself unable to put on paper in general terms.

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This being so I am forced to content myself in the first place with a review of the chief circumstances met with in cases of acute abdominal disease, attempting, so far as I can, to assess the value of each as an indication for operation in the majority of cases. In the second place I propose to refer briefly to cases of the most common disease that belongs to the group under consideration, namely appendicitis, indicating the views I hold with regard to the question of operation.

In considering the various points I shall adhere as much as possible to the order in which they will be met with in examining the patient.

(1) *Previous history.*—This may be very important. There may have been previous attacks as in appendicitis, or there may have been symptoms pointing to gastric or duodenal ulceration, gall-stones or renal calculus. Or immediately before the present illness unsuitable food may have been taken, suggesting the possibility of the case being one of acute gastritis, enteritis, colitis, or ptomaine poisoning. Again, the occupation may bring to mind the possibility of lead colic. Finally, there may have been symptoms pointing to tabes dorsalis and suggesting the possibility of the present attack being a gastric or intestinal crisis.

(2) *Mode of onset.*—In practically all cases this is acute and is associated with abdominal pain, and has, therefore, little practical value as an aid in recognising surgical conditions.

(3) *Vomiting.*—This is so constant in the early stages of many diseases that its mere presence at the commencement of an acute condition is of no great significance. If, however, it is a persistent feature for more than twenty-four hours, it constitutes strong evidence of an acute abdominal lesion, such as obstruction or peritonitis, and is thus a definite indication for surgical interference if accompanied by other signs pointing in the same direction. On the other hand, entire absence of vomiting is so rare in severe abdominal lesions that it should weigh *against* immediate operation in an otherwise doubtful case.

Recurrence of vomiting later in a case after its temporary cessation is, however, a *very* important sign, and may alone indicate very strongly the necessity for operation. For instance, a case of apparently mild appendicitis is seen and is not considered to be one requiring operation. After doing well for a few days a sudden attack of vomiting occurs. This almost certainly means perforation of the appendix or spread of peritonitis previously localised, and is frequently the first sign of one of these two conditions. Vomiting under these circumstances is then a most *definite* indication for immediate surgical interference. On numerous occasions I have seen cases in which recurrent vomiting has occurred in this way. I have always operated at once, and I have never regretted doing so.

(4) *State of the bowels.*—Absolute constipation both as regards fæces and flatus from the time of onset is certainly an important sign of either obstruction or peritonitis. Doubt on this point should be cleared up by the administration of a large enema. Return of the enema with some flatus and a little fæces certainly does *not* eliminate intestinal obstruction as it is frequently erroneously thought to do, but by itself is of little value. If, however, the enema is retained, either acute obstruction or peritonitis with resulting paralysis of the bowel is almost certainly present, and in the majority of cases operation is indicated.

On the other hand the absence of constipation is of no particular significance, for in the early stages of many cases of peritonitis and obstruction one or more actions are not uncommon, and in a few cases of peritonitis diarrhœa may be a feature. As an instance of this I might mention the case of a child who was admitted last summer into the Children's Hospital, Great Ormond Street for acute diarrhœa and vomiting. This was for two days looked upon as ordinary summer diarrhœa; then, however, the abdomen became distended and somewhat rigid, so I was asked by Dr. Garrod to see the case with him. I advised operation, and on opening the abdomen general pneumococcal peritonitis was found to be present.

(5) *The pulse.*—All will, I think, agree that in many of these cases the pulse is one of the most important guides that we have. In considering the pulse it will be useful, I think, to somewhat subdivide the cases into different classes. Taking first of all cases of acute intestinal obstruction, here the pulse as a general rule is an index of the duration and severity of the obstruction. In the early stages of acute obstruction in an otherwise healthy individual the pulse is not much affected, or is only to a slight degree increased in rate. This is pre-eminently of course the time for opera-

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tion; in other words, given the symptoms of acute intestinal obstruction, operation should be performed without delay, and we certainly should not wait till the pulse-rate has risen.

Later in the case, as the stage of collapse with rise in the specific gravity of the blood owing to loss of tissue fluids supervenes, the pulse-rate rapidly rises and its volume diminishes, the patient quickly passing into the condition when operation is well-nigh hopeless.

I should like to take this opportunity of strongly emphasising this point, for I am convinced that the present *deplorable* results of operation for acute obstruction are largely due to the waste of valuable time during which the patient is rapidly losing his tissue fluids.

The next class to be considered is that of acute perforations into the *general* peritoneal cavity. Here the pulse varies greatly according to the stage at which the patient is seen. Three definite stages may be recognised. The first is the stage of shock, due to the sudden escape of irritating fluid into the general peritoneal cavity. The pulse becomes small and very rapid, the amount of shock varying largely and being especially influenced by the type of patient and the quantity and quality of the material that reaches the peritoneal cavity. This is the stage during which operation should certainly *not* be performed, as the patient is almost certain to succumb to the additional shock caused by the operation itself. This stage in a few cases is so severe that it kills the patient outright. I have seen two such. One was a lady who had suffered for some years from symptoms of gastric ulcer. Perforation took place and she was seen an hour or two later by Dr. Hale White, and shortly afterwards by myself. She was almost pulseless from the shock and died half an hour after I reached the house.

The other case was an old man who fell down unconscious in the street. He was taken to the hospital and I saw him shortly afterwards. He was cold and nearly pulseless, and obviously had a perforation of some viscus. In spite of attempts to revive him he quickly died, and at the post-mortem was found to have a recently perforated duodenal ulcer.

After a varying period, usually a few hours, shock passes off and the second stage, namely reaction, succeeds it. The pulse now improves markedly in volume and dimishes in rate, as a rule, to about 100 per minute or a little less. At the same time the patient's general condition greatly improves, his colour returns, he becomes warmer, pain diminishes, and he feels in all respects better. This is the ideal moment for operation, and the results of surgical interference in this stage, which usually lasts a few hours only, are remarkably good. At the same time this, the best opportunity, is only too frequently lost owing to the great improvement in the patient's condition raising doubts as to the diagnosis.

As time passes the third stage of *collapse*, owing to loss of tissue fluids and toxæmia, due to absorption from the peritoneum, supervenes, and with this the pulse steadily rises in frequency and diminishes in volume. In the presence of other signs of peritonitis a pulse-rate of over 100 per minute at this stage constitutes an urgent indication for surgical interference, and of course the more rapid the pulse the stronger the indication. In less severe cases of peritonitis, such as those of appendicitis associated with local peritonitis, the stages above-mentioned are often not distinctly differentiated, at any rate, in so far as the first two stages are concerned. In such cases, however, the question of surgical interference will often arise after the illness has lasted for several days. For instance, either suppuration, perforation of the appendix, or spread of peritonitis may supervene. In both cases a rise in pulse-rate is the rule, and is to be looked upon as a most important guide when deciding whether operation should be performed. I recently operated upon a case which illustrates this point extremely well, and, moreover, shows how important it is to watch the pulse-rate closely in these cases.

The case was that of a man in Guy's Hospital under the care of Dr. Hale White. He had been admitted for an attack of appendicitis of forty-eight hours' duration. For two days after admission he did well and the attack appeared to be simple and local. Then, however, his pulse-rate suddenly shot up from 80 to 120 per minute, without any vomiting, or increase of pain or abdominal rigidity. I saw the man with Dr. Hale White, and suspecting that this sudden rise of pulse-rate was the first sign of perforation I advised immediate operation. On opening the abdomen I found a gangrenous appendix which had evidently just perforated. This I removed, and as the amount of

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peritonitis was slight I was able to completely close the abdominal wound two days later.

(6) *The respiration.*—In true abdominal cases the respirations as a rule are shallow and not to any degree increased in rapidity, so that if there be a marked *increase* in the respirations, especially if they are also of the jerky spasmodic type so frequently seen in pneumonia, and are accompanied by increased movements of the alæ nasi, great care must be taken to exclude the possibility of an early acute pneumonia or pleurisy being the cause of the abdominal symptoms.

The presence of marked pyrexia, cough and diminished air-entry at the bases will be of great assistance here, especially in differentiating early pneumonia from certain cases of perforation of a gastric ulcer in which the diaphragm is early affected. In these latter cases the respirations will usually be found to be rather jerky from the pain caused than actually rapid, and the temperature is usually subnormal or but little raised, as against the marked pyrexia of pneumonia.

(7) *The temperature.*—In intestinal obstruction and in severe perforative peritonitis the temperature, especially in early stages, is usually either normal or subnormal, and is seldom raised to any marked degree; in more local forms of peritonitis, on the other hand, the temperature is often raised several degrees. As a general rule, therefore, a rise in temperature in the early stages of an acute abdominal case is rather a point against immediate operation than for it.

(8) *The tongue.*—The tongue in many conditions becomes dry, but it seldom becomes so markedly and so rapidly dry as it does in severe cases of peritonitis and intestinal obstruction. In association with other symptoms and signs of peritonitis or obstruction a very dry tongue should therefore, I think, be looked upon as a decided point in favour of operation. On the other hand, a decidedly *moist* tongue should make one hesitate unless, of course, other points favouring operative interference are much in evidence.

(9) *The condition of the abdomen.*—In a very large majority of cases the question as to whether an abdomen should be opened is finally decided by the result of the examination of that abdomen, and in only a very small minority can the abdominal signs be definitely neglected in coming to a decision.

To take the second part of this statement first, one may safely say, I think, that the abdominal signs can only be neglected when they are negative. For instance, if there are no abdominal indications for operation and yet other indications are strong, an operation should in a very few cases be performed at once. But these cases are very rare, and as a general rule it is safer to wait a little while for the development of abdominal signs to confirm the diagnosis before operating. The case I have just referred to is, perhaps, a case in point, namely, the one in which I operated because the pulse-rate had suddenly risen, and in the absence of any abdominal signs showing that anything serious had occurred. Other cases are those which I mentioned under the head of "Vomiting," in which a patient with an apparently subsiding mild appendicitis is suddenly sick. This almost certainly, even in the absence of increased abdominal signs, means spread of peritonitis, and is a strong indication for operation.

To return to the majority of the cases, I propose to take in order the facts to be observed in abdominal examination. *Pain* need only be referred to to be dismissed, for although generally present it is so variable in degree, and in itself is certainly not an indication for operation, since it is present in greater or less degree in such a large number of conditions which do not call for surgical interference.

Distension of the abdomen is not present in the very early stages of either obstruction or peritonitis; its absence, therefore, certainly does not contra-indicate operation. On the other hand distension soon shows itself, so that although it certainly should not be waited for, its presence is more often than not an indication of a serious abdominal lesion.

Abdominal respiration, it may quite safely be said, I think, is *always diminished* or *absent* if peritonitis is present. The absence of movement may be local and affect only the upper or the lower part of the abdominal wall, and in this way positively indicates the position and extent of the peritonitis. Should, however, the normal respiratory movement of the abdominal wall be entirely absent, this alone constitutes very definite evidence of a general peritoneal lesion, and is, perhaps, one of the strongest possible indications for immediate surgical interference.

It must be remembered, however, that the

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converse is not true, for in most cases of acute obstruction the movements of the abdomen are not affected, at any rate in the early stages when operation is most strongly indicated. Later, as peritonitis supervenes on the obstruction the mobility of the abdomen is necessarily diminished. It may be said then that in any acute abdominal case the absence of the normal respiratory movement of the abdominal wall is a definite indication for surgical interference, but that the presence of the normal movement is no contra-indication.

Rigidity of the abdominal wall goes with absence of the respiratory movement, and is an equally important guide. Perhaps it would be more correct to say that it is more important, for in some cases the abdomen moves with respiration to some extent, but on careful palpation is found to be more or less rigid. This must be looked upon as a definite sign of peritonitis, because it is seldom absent altogether if there *is* peritonitis, and is, as far as I know, not present in the *absence* of peritonitis. Moreover, abdominal rigidity may, under certain circumstances, be the *only* reliable guide as to the necessity for operation. I refer to cases in which opium or morphine has been administered. In these cases the whole aspect of the patient, and all the signs but one, may be altered in the direction of disguising the importance of the case. The one sign that is not affected by opium and morphine is the one under discussion, namely abdominal rigidity; this, thank Heaven, is not altered to any appreciable extent. A rigid abdomen, therefore, is a definite indication of peritonitis, especially if opium or morphine has been given.

It may be here noted that in some cases of thoracic inflammation a spurious abdominal rigidity is present. On palpating the abdomen the wall may, for a moment, feel absolutely hard, but relaxation of the muscles takes place during each sudden, jerky, expiratory movement. Such a condition indicates pneumonia or diaphragmatic pleurisy, and is quite distinct from the permanent rigidity associated with peritonitis.

Definite and maintained muscular rigidity is, then, an indication for surgical interference, but again the converse is not true, for in certain abdominal conditions requiring immediate operation—in particular acute intestinal obstruction—rigidity is absent, especially in early stages, before secondary peritonitis has developed.

Tenderness on palpation and percussion are, as a rule, marked signs of serious abdominal lesions, such as peritonitis, obstruction, and pancreatitis, whereas they are absent in other non-surgical affections of the abdomen, such as colic and gastritis. Moreover, the site of most marked tenderness is frequently a reliable guide to the site of the lesion, and so will be helpful in deciding the question of operation in a given case.

On the other hand, extreme tenderness and hyperæsthesia are present in certain quite unimportant abdominal affections, especially in neurotic women. I well remember being called to see a woman late one night at Guy's. She had been admitted with a pulse of 120 and a history of twenty-four hours' abdominal pain and severe vomiting. On examination the abdomen was found to be uniformly tender and hyperæsthetic, but it moved well on respiration, and was not really at all rigid. Moreover an enema had been followed by a good result. No operation was performed on account of the absence of muscular rigidity, and the patient quickly recovered. Definite cutaneous hyperæsthesia over the region of the appendix certainly frequently denotes an inflamed and distended appendix, and may, when present, be a useful guide.

Auscultation of the abdomen is, I think, not sufficiently practised, for it may give useful information which may help in deciding the question of operation. In severe general peritonitis, such as that due to perforation of a viscus, there is a marked absence of the normal sounds of peristalsis, no doubt owing to the paralytic condition of the intestines. In cases of obstruction, on the other hand, marked peristalsis will often be heard above and in the neighbourhood of the obstruction, and thus be of assistance in diagnosing and localising a mechanical obstruction or strangulation.

With regard to the absence of liver dulness, too much must not be made of this as a sign of free gas in the peritoneal cavity, for when free gas is present the liver dulness is not invariably lost, and, again, little or no liver dulness can be made out in some cases in which there is certainly no free gas present. The sign may, however, be a most important and helpful one in certain cases, namely, those in which definite liver dulness observed at one examination is found to be diminished or lost when a second examination is made a few hours

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later. Such an observation *certainly* denotes free gas in the peritoneal cavity, and is an indication for surgical interference.

(10) *Rectal examination.*—This should not be omitted in any doubtful case, for it frequently helps to localise the cause of symptoms otherwise obscure, intussusception, rectal growth, pelvic appendicitis and pelvic abscess being among the conditions that may be recognised in this way. To give one instance: I was asked to see a boy, æt. 14 years, who had a forty-eight hours' history of severe vomiting and pain in the lower part of the abdomen. The boy looked very ill, had a pulse of 120, was tender above the pubes but not rigid, and the abdomen moved well on respiration. So far the case was indefinite, but on rectal examination an enlarged tender appendix was easily felt lying in front of the rectum. On opening the abdomen the pelvis was found to be full of sero-purulent fluid, and hanging down into it was a perforated gangrenous appendix. This was removed and the boy rapidly convalesced. Here, then, the rectal examination was the decisive factor, and but for it the boy might have been allowed to develop a rapidly-spreading diffuse peritonitis.

(11) *Leucocyte count.*—In a few cases this may be of service. For instance, in a case of acute appendicitis in which a definite localised swelling is present, it is often very difficult to decide whether pus is present and operation needed. For a lump of considerable size does not necessarily mean pus, and if pus is not present is better left alone. In such cases a marked leucocytosis, say of 30,000–40,000 per c.mm., especially if repeated observations show it to be rising steadily, strongly indicates the formation of pus and therefore is an indication for operation.

(12) Lastly, I think I should emphasise the importance, especially in doubtful and anomalous cases, of a more general examination of the patient. Naturally I am unable here to enter into this matter in detail, so I will content myself with the mention of two instructive cases. The first was a man, æt. 40 years, upon whom I was asked to operate. For three days he had suffered from violent vomiting and severe abdominal pain. When I saw him he looked ill and sunken, his pulse was a little over 100 per minute, his tongue dry, the temperature subnormal, and his abdomen tender in the epigastric region. His bowels, however, had acted, and his abdomen moved well and was not rigid. It was clear then that he had neither complete obstruction nor peritonitis, and the question of some obscure condition such as pancreatitis arose. However, I then discovered that he had no knee-jerks, that he had suffered from lightning pains in his legs, and had Argyll-Robertson pupils. Needless to say no operation was performed, and the attack passed off in a few days.

The second case was a man admitted into Guy's Hospital under Dr. Newton Pitt. The day previously he had eaten several oranges, and soon afterwards was seized with abdominal pain and severe vomiting, which persistently continued long after he had got rid of his gastric contents, so that he was vomiting material from his upper intestine. He seemed ill, had a pulse of 125, was somewhat tender in the upper part of the abdomen, but movement was good and rigidity absent. A turpentine enema, moreover, brought away only a small amount of fæces and flatus. It was decided not to operate at once because his urine was found to contain albumen and casts, so that the condition might be uræmic. And so it turned out to be.

I realise that in what I have said I have somewhat exceeded my instructions, for in relating the two last cases, and in various other parts of this address, I have dealt with *contra*-indications rather than *indications* for operation. I trust, however, that I shall be excused for doing this, for I feel sure you must all realise how inseparable the two sides of the question are.

As appendicitis is of all acute abdominal conditions requiring operation by far the commonest, I propose now for a few minutes to deal with this condition separately and to indicate briefly what I believe to be the best plan to adopt with regard to the question of operation.

If the case is seen within forty-eight hours after the onset of the attack, in my opinion operation should at once be performed in all cases. In this way I am quite convinced that many valuable lives will be saved. If the operation is done during this period—the first forty-eight hours—the risk is a very small one, and the gain a very great one. For the appendix can always be removed and the wound, in nearly every case, completely closed at once. Thus the patient is placed in safety, the attack is cut short, and the possibility of a future attack eliminated. Moreover, we must

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remember that we cannot be absolutely certain of the condition of the appendix, and whatever the symptoms and signs may be, there is always a possibility that the appendix is sloughing and will shortly set up fatal peritonitis.

Again, should an abscess form later it will have to be drained, the appendix probably being left behind, so that after many painful dressings and a prolonged illness the patient will recover with either an actual or potential ventral hernia, and the liability to future attacks from the remains of his appendix. All this is avoided by operation within forty-eight hours of the onset of the attack. Should, however, the patient not be seen till after forty-eight hours have elapsed, the question of operation will depend upon whether a swelling is present, indicating that the mischief is localised and shut off by adhesions.

If a *localised swelling is present* no operation should be performed immediately, but the case should be watched for indications of pus formation. Then, later on, if an abscess does form it must be opened.

The chief indications of pus formation are:

(1) Steady increase in the size of the mass.
(2) Progressive rise of temperature.
(3) Progressive rise in leucocytosis.
(4) Increase of pain and tenderness.
(5) Œdema of the abdominal wall.

If, when the case is seen, there is *no* localised swelling, an operation should be performed unless the attack has obviously been a mild one, so that the patient is at this stage practically convalescent.

If, for some reason, it is decided not to operate, the chief signs that should lead to a revision of that decision are as follows:

(1) Rise of pulse-rate.
    Hourly observations should be taken so that any marked rise will be recognised at the earliest possible moment.
(2) Increase of tenderness.
(3) Increase of rigidity.
(4) Reappearance of vomiting.
(5) Sudden fall in temperature, if associated with a rise in the pulse-rate.

The appearance of any of these changes while the patient is lying in bed almost certainly means either perforation of the appendix or spread of peritonitis, and hence should be regarded as a definite indication for immediate operation.

February 24th, 1908.

PNEUMOCOCCIC PERITONITIS.*

By H. D. ROLLESTON, M.D., F.R.C.P.,
Senior Physician to St. George's Hospital; Physician to the Victoria Hospital for Children.

PNEUMOCOCCIC peritonitis is well recognised but is rare. Netter only found it twice in 140 cases examined bacteriologically, and at the Victoria Hospital for Children Dr. J. D. Marshall, the Resident Medical Officer, has only been able to find one case at all likely to be pneumococcic peritonitis during the last eight years. On the other hand, at the recent discussion at the Royal Society of Medicine, Gossage reported four cases of primary pneumococcic peritonitis without pneumonia, all under 3 months of age and all fatal; these cases were from the East London Hospital for Children, where Bowen and Annand worked specially on this subject.

Though rare, pneumococcic peritonitis has attracted a good deal of interest on account of its peculiar incidence, and because in probably half the cases it is primary, and so accounts for a considerable proportion of the rare cases of primary peritonitis. It is commoner in children than in adults, and in Bowen and Annand's ninety-one cases under fifteen years of age it was commoner in girls than in boys in the proportion of three to one; after that age the incidence in the sexes is equal. From its greater frequency in young girls it has been thought that it may gain an entrance through the Fallopian tubes; against this it has been argued that there is generally no evidence of vulvo-vaginitis either clinically or at autopsy. On the other hand, Bond's observations on ascending mucous currents in the uterus and tubes make it probable that the pneumococci can be rapidly carried up before there is sufficient time for local inflammation to develop. A good deal of discussion has taken place as to the means by which pneumococci get into the peritoneal cavity; they may be brought by the blood-stream from foci elsewhere, such as the throat or lungs. Pneumococcic peritonitis is rare in pneumonia; thus, in 4454 cases of pneumonia obtained by adding together the statistics contributed by Hadley, Pasteur, Gossage, and Owen, to the recent dis-

* Delivered before the Medical Society of London.

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cussion at the Royal Society of Medicine, peritonitis occurred in eleven fatal cases or ·24 per cent. Pneumococcic peritonitis may also be secondary to pneumococcic gastro-enteritis or appendicitis. Pneumococcic polyorromenitis—the pleuræ, pericardium, and peritoneum all being infected—may occur especially in children. In about half the cases the peritonitis is encysted. As in tuberculous peritonitis the abscess may open externally at the umbilicus. Inasmuch as it often begins low down in the abdomen it is very likely to imitate appendicitis, and extremely difficult to distinguish from it. Encysted peritoneal abscess may supervene after diffuse pneumococcic peritonitis; thus in a case under my care in St. George's Hospital, signs of a bilateral subphrenic abscess, first on the right side and then on the left, followed drainage of pelvic pneumococcic peritonitis. The abscess on the right side was opened by my colleague, Mr. Pendlebury, who, however, thought it wiser not to interfere with the left side, and in this he was, as it turned out, fully justified, for gradually the signs and symptoms pointing to a left-sided subphrenic abscess disappeared. This raises the question whether, as in pneumonia, the exudation of pneumococcic peritonitis may undergo autolysis and be absorbed. The disease may be divided into three stages: first there is an acute onset resembling appendicitis; then a period in which the symptoms subside, but diarrhœa being usually present, typhoid fever may be imitated; and thirdly a prolonged stage in which residual abscesses may form, especially in the upper part of the abdomen, the condition resembling tuberculous peritonitis.

February 24th, 1908.

Tuberculous Disease of the Joints.—It makes no difference what the joint is; if it is the subject of tuberculous disease it must be kept at rest. Plaster-of-Paris and other splints: splints made of common house-flannel, hardened by plaster-of-Paris, are very useful in the treatment of tuberculous joints. The flannel having been duly cut into shapes and laid ready, an inch or so of water is poured into a basin, and fresh plaster is shaken in until it forms a little island in the middle; then it is quickly stirred in until a creamy mixture is made with which the flannel is to be soaked. It is well to have two pieces of shaped flannel on each side of the limb, one of which has a surface daubed with the cream on one side only, the dry side being placed next to the bare limb, and on the outside of this is laid the piece which is thoroughly soaked with the cream. A nurse, or other assistant, holds these two pieces close against one side of the limb, whilst the surgeon quickly fixes them in place by a few turns of a muslin roller. Without any loss of time the other side of the limb is similarly treated, only in this case the splints are put on outside the muslin bandage which held the other pieces in place. If the splints have been put on nicely there will be a space running up the front of the limb between their borders, which is covered in simply by muslin, and then, or next day, the surgeon may run the scissors up this gap, take off the splints, trim their edges, and loosely re-apply them. But of all ways of fixing an inflamed joint there is none so good as that of leather side-splints. These splints are made of undressed cow-hide, shaped to a paper pattern, and soaked in hot water and vinegar. Thus treated, the leather becomes as limp as wet brown paper, and after being bandaged on the limb in two side-pieces it is left on for twelve or twenty hours to harden, and it may be afterwards lined with washleather, and fixed with straps and buckles.

The above is taken from an 'Index of Treatment,' edited by Robert Hutchison, M.D., F.R.C.P., and H. Stansfield Collier, F.R.C.S., and published by Messrs. John Wright & Co., of Bristol. The work provides the practitioner with a complete guide to treatment in moderate compass, and in a form convenient for reference. No pains have been spared to make the book a trustworthy index to the best and most modern methods of dealing with disease. Care has been taken to avoid embarrassing the reader with a large choice of procedures, and therefore those only have been described which, in the opinion of the respective writers, are considered the simplest and most effective. In view of the special audience to whom the book is addressed, no attempt has been made in the surgical articles to deal with the more elaborate operations; on the other hand, non-operative treatment has been dealt with in detail, as well as such minor or emergency operations as any practitioner may be called upon to perform.

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* *Specially reported for the Clinical Journal. Revised by the Author.*

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CARCINOMA OF THE BREAST: ITS EARLY RECOGNITION AND TREATMENT.*

By R. H. JOCELYN SWAN, M.S.Lond., F.R.C.S.Eng.,

Assistant Surgeon to the Cancer Hospital, Brompton.

GENTLEMEN,—The subject of cancer in all its forms is now occupying so much the public mind as well as the lay press that no apology is needed from me when I bring it before you by selecting cancer of the breast for this lecture. My reason lies in the fact that the condition is frequently so difficult to diagnose in an early stage from other mammary tumours. Doubtless if patients could be prevailed upon to seek advice as soon as they noticed any lump or pain in the breast, our operative results would be immensely improved, but unfortunately patients have such a dread of cancer, and they seem so afraid that such a verdict will be given if a doctor be consulted, that they are content to hide the tumour until they become alarmed by the increase in size or by the onset of acute pains. If only the general public could be educated to the fact that cancer can in a very large number of cases be entirely eradicated, if the case is seen in an early stage, I feel sure that a very great advance would be made, an advance which has doubtless been temporarily delayed by the premature effusions in the lay press. Much work has recently been carried out upon the spread of cancer of the breast, and the adaptation of these to our methods during an operation should ultimately increase the proportion of patients who remain free from recurrence.

Cancer of the breast forms about one half of all cases of mammary tumour, and with uterine cancer is the most common form of cancer in women. It usually occurs between the ages of thirty-five and fifty, although occasionally we find a younger

* A Lecture delivered at the Medical Graduates' Polyclinic.

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patient affected. Personally, the youngest patient I have seen with mammary carcinoma was æt. 28 years, in whom a very rapidly-growing tumour was present in a lactating breast, and it may be taken as a general rule that when cancer occurs in a comparatively young patient it forms a rapidly-growing tumour with marked malignancy and with early dissemination. Mammary carcinoma occurs occasionally in the male breast, between 1 and 2 per cent of mammary carcinomata being found in the male.

Cancer may occur in any part of the breast. Perhaps it is most frequently situated in the upper and outer quadrant, rather nearer the nipple than the periphery, but it may be placed deeply embedded in the breast tissues or in an outlying nodule at the extreme periphery of the gland. Increase in size of the tumour takes place by the gradual infiltration of the growth, but the most important spread is that which takes place by the lymphatic channels. The arrangement of these lymphatics draining the mammary gland is exceedingly important, not so much from their anatomical relations, but by showing the route taken in the dissemination of the disease and also the extent that has to be removed during an operation to get clear of any possible source of recurrence. So important are these that I propose to spend a few moments to run over this lymphatic arrangement.

The great majority of the lymphatics from the gland tissue pass into a central subcutaneous plexus (the *subareolar plexus*), from which the efferent lymphatic channels are usually two, reinforced by smaller, which pass along the lower pectoral border, and perforating the axillary fascia end in the thoracic chain of axillary glands.

The *accessory channels* are: (A) a *subclavian vessel* which drains the deep aspect of the breast, perforates the pectoralis major, and, running above the pectoralis minor, terminates in a gland to the inner side of axillary vein.

(B) *Internal mammary vessels* with the perforating arteries, pass from inner aspect of breast to the retro-sternal glands along internal mammary artery.

Some recent work by Handley has had a most far-reaching effect in explaining the spread of cancer from the breast. Handley has shown that the spread takes place peripherally by permeation of the lymphatics by cancer-cells; that this spread may be in part downwards and inwards along the lymphatics on the sheath of the rectus muscle, and thence, perforating the cellular interval between the two muscles, pass directly into the triangular ligament of the liver, and gain entrance to the portal glands and abdominal viscera.

Further than this, it has been shown that lymphatic vessels run along and anastomose freely on the deep fascia covering both the pectoralis major and the serratus magnus muscle, and in consequence this must be widely removed during any radical operation on a case of mammary carcinoma.

Causation.—The subject of causation of cancer is far too long to enter upon here, but there are several points in connection with mammary growths that I would like to mention. First, what is the influence of previous inflammation in the breast to the production of cancer? This is a very difficult question to answer. I have seen several cases in which a very small nodule of carcinoma has been found on section to be embedded in a mass of chronic mastitis, but this by no means proves that all growths commence in the same way. If we take 100 consecutive cases of mammary carcinoma, we find about 75 per cent. occur in married women in whom inflammatory changes in the breast are so common; but are not a similar proportion of women married before they reach the cancer age? Again, only about 30 per cent. give a history of previous inflammations. Now in the woman of forty there must always be areas of damaged tissue in an organ that has gone through so many changes during the child-bearing period, and we know full well the liability of cancer to develop on damaged epithelial tissues, as in the tongue; so that the two may be analogous and explain the frequency with which cancer develops in the breast, but *not* the cause. How often we hear that a tumour has been found in the breast following upon a blow or some injury. This is true, but is it cause and effect? I would say "No," for almost any woman will remember a blow on the breast, to which she will attribute the lump she has subsequently found.

The clinical course of cancer of the mamma varies within wide limits. Commencing in a lobule of breast tissue, probably from the epithelial lining of an acinus, the cells infiltrate through the wall into the lymphatic spaces. Progressive infiltration from the peripheral portions of the growth with gradual permeation of the lymphatic channels continue until the patient's attention is drawn by some

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symptom to her breast or until a tumour is found by accident. I have heard several patients tell me they found a lump whilst washing. The increase in size of a carcinoma of the breast varies enormously, some being remarkably malignant with rapid dissemination, especially in young patients or in a breast lactating; others are very slow, taking years to reach the skin and forming the well-known atrophic form. All grades intermediate between these are met with, the slower forms containing more fibrous tissue, which merited the old name of "scirrhus." When the tumour is first discovered it is usually the size of a small cherry, very firm, but with no defined limitations, gradually fading into the breast tissue, and often nodular. The skin over it is at first freely movable, but with the steady progress of the disease the fibrous bands connecting the breast tissue to the skin—the so-called "suspensory ligaments" of Astley Cooper—become drawn upon, and although the skin is not actually infiltrated, it *becomes dimpled* over the surface of the tumour. If the surrounding mammary tissue be pinched up the skin over the mass is found to be less free than elsewhere, and assumes a pitted form which has been likened most aptly to the peel of an orange. This is very distinctive, and I know of no other mammary affection which will produce the same effect. Later the skin becomes actually adherent to the underlying tumour, reddened or livid in colour, and ultimately ulcerates.

If the tumour be placed in a more central position the lymphatic spaces around the lactiferous ducts become infiltrated, and by the contraction of these the nipple becomes *retracted*. This symptom has been made too much of, for if the growth be one which is progressing rapidly no retraction will be found, even in a centrally-placed tumour, whereas if it be at the periphery the nipple may remain unaffected. A point may be mentioned, however, which was first brought to my notice by my colleague, Mr. Charles Ryall. If the tumour be situated away from the nipple, pressure upon the space between the mass and the nipple will often produce some drawing-in of the latter, which does not occur if the tumour be an innocent one or a cyst.

At the same time it should be remarked that retraction of the nipple may be present from a congenital malformation, or from old chronic inflammations or abscess, and also that a deeply-situated growth, especially in a large breast, may produce neither dimpling of the skin nor retraction of the nipple.

Pain is often given as an early symptom of carcinoma of the breast, but if we were to rely upon this symptom we should often be grievously misled. In my experience pain in mammary cancer is extremely variable—in fact, I have seen one case so advanced that the patient was admitted into hospital with a spontaneous fracture of the femur from a metastatic deposit, and it was only on a thorough examination of her that the primary source was found to be a carcinoma of the breast which was entirely unknown to the patient, and had given rise to no pain whatever. I could give you many cases in which pain has been entirely absent, and I find this usually so in stout women. There is, however, one form of pain to which we must place serious import, and it is one which often leads a patient to examine her own breast. I allude to a sharp twinge or pricking pain which is felt in the breast and in the thorax, sometimes in the arm, and I would ask you never to omit to examine the breast of a patient complaining of these sharp stabbing pains in the thorax.

Local pain in a cancer of the breast is exceptional unless the skin is already involved, so that if pain be caused upon pressure on a mammary tumour we suspect a deep-seated abscess or inflammatory process rather than a new growth.

Another sensory effect I have frequently noted is that of numbness or of "pins and needles" in the fingers, but I am of opinion that this only occurs from pressure on the nerves by the enlarged axillary glands, and is thus a symptom of a later stage of the disease.

And now a word about the physical examination of a breast the seat of a mammary tumour. In the first place no tumour should ever be examined by picking it up between the fingers, but rather by the flat of the hand, and I find it always a good practice to examine the breast whilst standing behind the patient. This is most important, for if a tumour still be felt with the flat of the hand, it shows at once that there is something more than an inflammatory mass, which may be a neoplasm, an innocent tumour, or a cyst, and it is between these that a diagnosis has often to be made in an early case. Feeling the mass under the fingers in this

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manner, an opinion can be formed of its hardness, of its limitation, and of its mobility in the breast tissue. *A firm, hard mass, incorporated in the breast tissue with no marked limitation, but gradually fading into normal mammary gland tissue and immobile, is almost certainly a carcinoma.*

In stout breasts, however, it is often very difficult to appreciate the mobility or lack of it, of a tumour, and it is here that error may be made. However, a suspicious dimple of the skin due to the contraction and pull of the suspensory ligaments is often present in cancer. Perhaps the greatest difficulty is met with in the diagnosis of cancer from a small tense cyst which is embedded in a mass of chronic mastitis in a fairly full breast. Here a firm, hard tumour will be felt, of which the outline is not defined and immobile. Firm pressure may give an impression of elasticity or may produce a little blood-stained discharge from the nipple, both of which are suggestive of cystic formation, but I can assure you that it is often impossible to be certain of the diagnosis in these cases. Should more than one tumour be present in the breast, cancer is unlikely, but it must be remembered that cancer may be present at the same time as a cyst or an adenoma.

Mobility in pectoral fascia.—Whilst examining a mammary tumour it is important to know whether the growth has already become fixed to the pectoral fascia, especially in one situated deep in the breast. To ascertain this the arm should be held up against resistance so as to place the pectoral muscle in action and the degree of mobility of the tumour *in the direction* of the muscular fibres noted; limitation of movement in this plane denotes involvement of the fascia, whereas movement across the direction of the muscle-fibres will occur with the fascia extensively involved.

Finally a search should be made over the mammary area for any subcutaneous nodule of growth, and in the lymphatic area for enlargement of the glands. The arm should be held slightly away from the side and examination by the flat of the hand made along the pectoral border and in the axilla. Firm, hard, enlarged glands may be felt, and if so are strong evidence that the mammary tumour is malignant, but in many cases no enlargement can be felt, but yet at an operation numerous glands are found to be infiltrated with carcinoma. The existence of glandular infection is not so important now that it is made a routine practice to remove the glands with the breast and lymphatic tract, but may help in arriving at a diagnosis. Finally the supra-clavicular area should be examined for infected glands.

Glands may be found enlarged in the axilla in other conditions than carcinoma. Thus they are enlarged, but tender in acute mastitis, mammary abscess or tuberculosis, and in cutaneous affections of the areola or breast, as eczema or syphilitic chancre. They are occasionally enlarged with chronic simple mastitis.

I have given you here the usual symptoms typical of carcinoma of the breast in its early stages, and if you asked me the symptoms upon which I place most credence, I should say the hardness of the tumour, the immobility in the mammary tissues and the dimpling of the skin over it. Still, the more one sees of these mammary tumours—and we have a great number sent up to the Cancer Hospital for a diagnosis—the more one appreciates the difficulty in diagnosing carcinoma in an early stage. I have frequently been very puzzled with a case presenting a small, hard, simple, immobile tumour in the breast in a patient æt. about 40 years, and have been quite unable to say whether it was a cyst or a carcinoma, and I well remember demonstrating in my clinic such a case as one of typical carcinoma (and it had been similarly pronounced by another surgeon), yet to find it was a very thick-walled chronic abscess on making an exploratory incision. If we can find any elasticity in the mass or any other swelling in the same or the other breast, we should always suggest a cystic formation in some chronic mastitis, but at the same time we must remember that carcinoma may start in a mass of mastitis, and that carcinoma may be present at the same time as a cyst. You will ask how are we to tell the difference and to be perfectly honest. I think the only way is by *exploratory incision.* Certainly it is by no means a confession of ignorance to have to advise this, but rather to be on the side of caution, for if carcinoma be found it gives the patient the very best chance of success by an early removal of the disease.

A simple fibro-adenoma is hardly likely to be mistaken for carcinoma. It occurs at a much earlier age, its limits are sharply defined, and it is very movable in the breast tissues. Still, the

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rapidity with which this innocent tumour may grow, especially in an active breast, may sometimes give a suspicion of malignant disease; a careful physical examination will usually suffice.

There is another and more rare lesion in the breast that I have seen mistaken for carcinoma, and that is a *tuberculous mastitis*. This forms a diffuse, irregular swelling in the mamma, causing some aching pain and gradually extending. There is no true dimpling of the skin, but the axillary glands may be enlarged, rather soft and tender. Usually in these cases I have been able to feel more than one mass in the breast, and they have occurred at an earlier period of life. In this specimen we had an important guide to our diagnosis in a typical tuberculous ulcer of the skin covering the mass, for the tendency of tuberculous disease is to caseate and break down, perforating the skin and leaving a troublesome ulcer discharging watery pus and bounded by thin, undermined edges.

Sarcoma of the breast occasionally occurs and may cause some difficulty. It occurs at any age, usually over thirty, forming an ill-defined, soft, rapidly-growing tumour. It is often slightly movable in the breast tissues and presents quite a different feel to the hand than a carcinoma. The nipple is not retracted nor is the skin dimpled, the growth is liable to sudden increase in size with pain due to hæmorrhage taking place in it.

Treatment.—And now as regards the treatment of these cases; and in the first place I wish to add a little to what I have said about exploratory incision. Suppose we are confronted with a lady over thirty-five showing a single, small, immobile and hard tumour in her breast, which she had just discovered either by accident or from a small pricking pain in it. We can find no other mass in the breast, it is somewhat deeply situated in, perhaps, a fat organ, the skin is not dimpled, the nipple not retracted, nor are any enlarged glands to be found in the axilla. We can get no sense of elasticity in the mass, but yet it feels smooth and rather like a cyst. How are we to advise our patient? I have purposely drawn out a case such as we constantly meet in practice, and upon which it is extremely difficult to say whether we are dealing with a cyst or a malignant growth. The more of these cases one sees the more one realises the difficulty of giving a definite opinion. If we tell our patient it is merely a cyst which will cause no harm by leaving it, we get precious little credit when later she returns with the tumour double the size and other symptoms of an obvious carcinoma, whereas if we wait and watch the progress to make more certain we run a very grave risk of allowing dissemination to occur. Even if we take the patient into our confidence and tell her we fear she may have cancer, she will go about in mental dread of the disease and suffer much misery. No; I think the wiser course is to explain the position most fully both to the patient and her friends, explaining the possibility of growth and the danger of leaving it until the diagnosis is obvious, and advise her to undergo a small exploratory operation to ascertain the true nature of the disease. I have never regretted having taken this precaution, and in this way have been able to remove several cancerous breasts in a very early stage with almost assured success, and on the other hand, have found some small tense cysts, removing them with immense relief to the mind of my patient. To make an exploratory incision, I repeat, is no confession of ignorance, but frequently a great aid to a true diagnosis and means of ensuring an operation in an early stage of the disease.

The incision should be made freely into the mass and the edges turned back so that the lump is brought directly into view. The feel to the knife and the appearance to the eye is distinctive, and no error should be made between growth and mastitis, whilst should any doubt exist a small piece can be removed for a microscopical examination. All bleeding is arrested and the wound accurately sutured. Assuming that the incision proves a cancer to be present, should we at once go on to the major operation and remove the breast, provided we have permission so to do? It has been shown by clinical experience that local infection readily takes place when a growth has been incised *in situ*, but if we realise this there ought to be no risk, and certainly we save the patient a second administration of an anæsthetic. Provided the incision is accurately and tightly sutured, that all instruments, towels and swabs are removed and replaced by fresh ones, and the surgeon and his assistant thoroughly wash up as if commencing the operation, I do not think any risk will accrue, and I have yet seen no reason why I should not go straight on. Personally I always

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wear sterilised rubber gloves for the exploratory incision, and change them with the other things if I go on to the major operation.

The operation of amputation of the breast for growth is no light one and aims not only at the removal of the breast but also of the lymphatic glands, draining the area as well as the lymphatic channels between the two. If we revert for a moment to the anatomy of the lymphatic system of the mammary gland, you will remember we spoke of the subareolar plexus into which the majority of the collecting lymphatics open. From this plexus we have the principal lymphatic channels passing along the pectoral border to the thoracic group of axillary glands. Then there is the subclavian accessory vessel, perforating the pectoralis major and passing above the upper border of the pectoralis minor to some glands on the inner side of the axillary vein, as well as the channels passing from the inner aspect of the gland with the internal mammary vessels to the retro-sternal group. I also mentioned Handley's work on the peripheral spread and the intimate lymphatic anastomosis on the fascia covering the pectoralis major, the serratus magnus muscles, and on the aponeurosis of the rectus muscle, necessitating a wide removal of the superficial and deep fascia. It is highly necessary to remove the whole area of possible lymphatic infection with the breast, and to do this in continuity so that in no place are infected lymphatics incised, for otherwise small foci may be left from which recurrence of the disease may commence. This can be performed at one operation, but each case must be judged on its own merits, and no stereotyped rules for the lines of incision can be laid down. These will vary with the position of the tumour, whether in the upper, lower, inner, or outer part of the breast, but the same general principles hold good. Incisions must be carried to enclose not only the growth but any area of possibly infected skin. There should be no attempt to save any suspicious cutaneous area so as to ensure closure of the wound afterwards, but rather to make certain that the disease is completely removed, even if a part of the wound remains unclosed. This can always be grafted later. The incision then is made in an elliptical manner around the breast, prolonged upwards so that the tissues in the axilla may be fully exposed. Personally I would make a strong point of this upward prolongation. I take it just above and internal to the pectoral border, passing up in to the anterior margin of the deltoid muscle and then curving downwards in the interval between the deltoid and pectoral muscles. Made in this way my ultimate scar does not pass into the axilla, but remains completely internal and above it, and I am satisfied that this position allows of a very much better movement of the arm. I have some cases waiting to show you and will demonstrate my point then. I now prolong my incision downwards further than formerly, especially if the growth is in a lower quadrant of the breast, so that I can remove the aponeurosis over the upper part of the rectus muscle in view of Handley's work on the spread of the disease. These incisions are made with one sweep, the lower one first, and then extensive flaps are made of the skin and small layer of fat, carrying them first downwards over the side of the thorax and then inwards as far as the mid-line of the sternum and as far upwards as the clavicle, so that no outlying lobule of breast tissue is left behind, and so that a much larger area of fascia can be removed than skin. I next incise the superficial fascia over the front of the sternum and costal cartilages, and detach the whole of the sterno-costal origin of the pectoralis major, picking up each perforating vessel as it is divided. Continuing this downward the anterior aponeurosis of the sheath of the rectus is removed, whilst above the fascia is divided so as to expose the separation between the sternal and clavicular portions of the pectoral muscle. The two portions are separated with the handle of the knife as far as the humeral attachment, which is then divided close to the bone. This large mass of breast, superficial and deep fascia, sterno-costal portion of the pectoralis major and the rectal aponeurosis is then raised from the inner aspect until the pectoralis minor muscle is fully exposed and the lower margin of this latter defined, the branches of the acromio-thoracic artery being caught up in forceps. This muscle is then raised, divided close to the coracoid process, and detached from its costal origin. I now leave this part of the wound, all the bleeding being arrested and the surface covered with a sterile towel wrung out of hot saline solution, and commence to remove the axillary tissues. First a few touches of the knife exposes the axillary vein close to the divided tendon of the pectoralis major muscle and the axillary sheath is opened. The vein is traced upwards as far as the clavicle, any tributary being caught between forceps and divided. In this way

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the axillary sheath is removed and turned down from above, and the dissection continued by removing the tongue-shaped piece of fascia from under the costo-coracoid membrane. This is gradually removed downwards into the axilla, the fascia being removed from the serratus magnus muscle at the same time with the thoracic group of lymphatic glands. The intercosto-humeral nerve is cut short, and continuing outwards the subscapular muscle is exposed. Care should be taken to preserve, if possible, the subscapular nerves, and this can usually be done, but tributaries of the subscapular vein require ligature. Finally a few strokes with the knife separates the whole mass from the latissimus dorsi muscle. The surface exposed is kept covered by hot saline cloths during the steps of the operation, which in this way removes the whole breast and surrounding superficial fascia, the rectal aponeurosis, pectoral muscles and fascia, and the axillary contents as far as the clavicle in one united piece without incising any infected lymphatic area. Finally the vessels are ligatured and the wound sewn up with thread, with a drainage-tube through the lower flap into the axilla. There is usually little difficulty in bringing the skin flaps together owing to the extensive undermining and to the amount of tissue removed.

Doubtless you will ask me whether it is necessary to remove so much in every case. To this I would answer "Yes." Even if I can feel no enlargement of the axillary lymphatic glands clinically, yet I know they are frequently infected, and thus must be removed in any attempt at a radical operation. Now is it necessary to remove the pectoral muscles, and what is the ultimate result as regards movement of the arm? First as to the removal: perhaps in a very early growth in the superficial portion of the breast it may be unnecessary to remove the whole muscle, so far as local infection is concerned, provided the pectoral fascia is thoroughly removed, together with some superficial part of the muscle, but in a deeper situated growth the removal of the muscle is essential, whilst in any case it allows of a much more satisfactory clearance of the axilla. With regard to the pectoralis minor I remove it for a different reason. To effectually clear out the axilla all the fascia on the deep aspect of the pectoralis minor must be removed, together with the triangular portion contained between the upper border of the muscle, the axillary vein, and the thoracic wall. To do this the anterior thoracic nerves supplying the pectorals are divided, so that if we leave the muscle it is useless, whereas its removal greatly simplifies our dissection and renders the operation more efficient. I do not think any axilla can be effectually cleared by raising the pectorals with retractors, and I always lay particular stress on the tissues beneath and above the pectoralis minor, becouse I find that so many cases which are sent to us at the Cancer Hospital for recurrence after removal of the breast for carcinoma show infection of this infra-clavicular group of glands, which have remained untouched at the primary operation. I am perfectly aware that this seems a drastic measure for a small mammary cancer, but I am sure that if a complete operation were performed in every case we should hear less of recurrence of the disease.

With regard to the movements of the arm after the operation, the removal of the pectorals seems to make very little difference. I make it a rule in my practice to keep my incision well away from the axilla, as I have previously explained, and I only keep the arm bound to the side for about thirty-six hours after the operation, after which the patient is encouraged to move the limb for herself, as well as passive movements being applied after the first week. As soon as the stitches are removed I have the shoulder and thoracic areas massaged daily to prevent adhesion of the incision to the underlying tissues, and usually the patient can after three weeks raise the arm and hand well. The only movement in which I had heard patients fail is that of pushing, as in sweeping.

If I have not already occupied too much of your time, I would like to show you some patients whom I have asked to come up to-day to show you the results I have obtained from this extensive operation, and I will ask you to examine them afterwards and see for yourself the truth of my statements. All but one of these women have had both pectorals removed at the operation, and you will observe the very excellent movement of the arm that each patient enjoys. In one of these cases of more extensive disease, I divided the clavicle at the same time, clearing out infected glands in the supra-clavicular and deep cervical chain. She remains well after some eighteen months, but in this case her movements are not so good. I bring these cases to-day in various intervals after their operation as they presented themselves at my out-patient department last week, in no way selected and not as cases to show cures, as sufficient time has not yet elapsed.

In conclusion, gentlemen, I must thank you for bearing with me so patiently in such a lengthy lecture, but if these remarks will serve to help any of you in the diagnosis of a doubtful mammary tumour, I shall be more than satisfied.

[Six cases were then exhibited to show the results obtained in the complete operation of removal of both pectorals as advocated above. The advantage of keeping the scar of the incision clear of the axilla was demonstrated, together with the very free movements in the arm enjoyed by each patient. Numerous pathological specimens from the Museum of the Cancer Hospital were also shown to demonstrate some of the difficulties in the diagnosis of mammary tumours.]

March 2nd, 1908.

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A DEMONSTRATION OF CASES OF BRACHIAL NEURITIS.*

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THIS afternoon I want to discuss brachial neuritis. I had a case of this condition at the hospital the other day which I think shows the symptoms of it very well; but before we talk about the cases of acute brachial neuritis I would like to sketch out the various forms of inflammation and injury to the nerves of the upper limb. For convenience I will put the cases into four groups: (1) traumatic, (2) pressure, (3) chill, (4) toxæmic. First, traumatic lesions of the nerves and roots. What sort of injuries are these? First, injuries to the roots themselves, that is to say of the primary nerves forming the plexus. The chief nerve to be injured is the fifth cervical, and when this is injured it is known as Erb's palsy. For instance, a man falls out of a cart three or four feet on to the tip of his shoulder, and in the effort to save his head he pulls it away from the shoulder, and in consequence the highest and longest root gets stretched, and in consequence there is a lesion of the fifth cervical root. The muscles specially affected are the deltoid, the biceps, the supinators, and the two spinati, the infra- and supra-spinati. The result of that will be paralysis, so that the patient cannot abduct his limb or externally rotate it. The deltoid and the biceps, which are the two most important muscles in the upper extremity, being paralysed, the arm is almost useless. That is a very common injury. You find the same thing in newly-born children, and it is then known as the Erb-Duchenne palsy, caused by the traction on the upper extremity in the act of birth. Various other primary nerves or divisions of the plexus may be damaged as the result of a fall on the arm. I have a case which I will show you directly to illustrate the results of damage to one of the mixed nerves in consequence of a fall on the shoulder. We will see if we can locate the lesion. There are many other forms. Dislocation of the shoulder is a very common cause of injury to nerves in the upper extremity. The nerve which especially gets damaged is the circumflex, a downward dislocation of the head of the humerus causing palsy, especially of the deltoid. With that muscle, the supra- and infra-spinatus often become paralysed, and the supra-scapular nerve must also share in the damage. Callus from dislocation of the elbow may cause paralysis. All sorts of injuries may cause palsy. One thing I would specially mention is the effect of muscular strain, which we will call a form of trauma. Muscular strain may cause damage to the limb—I mean the actual strain caused by the man's own effort. For instance, a powerful effort has been known to paralyse the musculo-spiral nerve, with the result that the man gets a wrist drop. Another form of paralysis which I saw the other day was due to a man working in a trench heaving up the dirt over his shoulder on to the surface. Usually when they have to work in a deep trench they have a platform half way and get it to the surface by two attempts, but here the man was, I suppose in order to save time, shovelling it directly on to the ground above. When putting up a particularly heavy shovelful he felt something give way in his shoulder, and at the same time experienced a stabbing pain, and he could not use his arm afterwards. Three or four weeks later I found he had paralysed his levator anguli scapulæ and rhomboid. He had some winging of his scapula, was unable to raise his shoulder-blade or to bring it back properly on the left side. There was some tenderness of the posterior scapular nerve, which on coming off from the fifth cervical root dips down through the muscles through the levator anguli scapulæ and ultimately reaches the rhomboid. This man, by this effort in digging and raising the dirt, had no doubt produced a traumatic neuritis. I have known a man paralyse the serratus magnus in the same way by a powerful effort, damaging the long thoracic nerve. That happened in a swimming race. He was using the side stroke, and after a powerful effort felt a stabbing pain at the back of his shoulder, and was unable to go on. He managed to get to the side and had to give up the attempt. A week later he had typical paralysis of the serratus magnus muscle with winged scapula, and no power of pushing forward the shoulder.

* At the Medical Graduates' College and Polyclinic.

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This woman, nearly a fortnight ago, fell off the lower step of a motor omnibus on to her shoulder. In falling she struck her head and cut her chin, becoming unconscious, and it was altogether a bad accident. When she came to, her arm was weak, and she is now unable to abduct the left arm. There is paralysis of the left deltoid, and the triceps is also paralysed, and you see that she cannot push the weight of her own forearm, and has dropped wrist. Her flexors are all right and her biceps is not paralysed, so it is not Erb's paralysis. These extensors of the finger and thumb and of the wrist are all supplied through the musculo-spiral from the posterior cord. The deltoid also is supplied ultimately from the posterior cord, by way of the circumflex nerve. Has she damaged her posterior cord or has she suffered a lesion of the circumflex nerve with a lesion of the musculo-spiral? To decide that, we must examine further to see if any of the other muscles which are supplied from the posterior cord are also paralysed. The circumflex nerve is given off just above the subscapulars, so if the posterior cord has been damaged by the fall the latissimus dorsi will be paralysed. To test the muscle, catch hold of the thick posterior fold of the axilla with the finger and thumb on each side, and make the person cough. In her case the right muscle contracts strongly, but the left does not. Clearly, then, she has damaged the posterior cord of the plexus.

The next group which I want to speak of are the *pressure palsies*. Of the causes of pressure on the various nerves and roots, I suppose the commonest is sleep, and we will include with that anæsthesia. Next pressure from a crutch, or compression by a tumour, or callus, though the latter is better grouped as traumatic; and lastly there is the pressure exerted by a splint or tight bandage. I have a case illustrating the results of tight bandaging to show you. Sleep palsies usually damage the musculo-spiral nerve. A man, perhaps in a drunken sleep on a Saturday night, hangs with his arm over the sharp back of a kitchen chair, and wakes up with paralysis of the musculo-spiral nerve. He comes up for examination perhaps on Sunday, perhaps on Monday morning, and for that reason these conditions have come to be known as Saturday night or Sunday paralysis. The result of this pressure is dropped wrist, and in this type the triceps usually escapes. He gets paralysis of the extensors of the fingers and wrist, and of the supinator longus. The reason is that the nerve is damaged below the point of exit of the nerves to the triceps. But crutch palsy more commonly includes the triceps in the paralysis, because the nerve is damaged above the exit of the nerves to the triceps. Otherwise the two conditions are the same. As a result of anæsthesia we have what is called post-anæsthetic paralysis, which is due to pressure on various nerves by the strained position in which the limb is placed during anæsthesia. It may be the musculo-spiral which is damaged, or other nerves. Sometimes the result of sleeping in an awkward position is to damage the ulnar nerve, with anæsthesia of the little finger and paralysis of all the interossei, and perhaps the flexor carpi ulnaris as well. The median nerve sometimes suffers, but not often, and then it will be from pressure across the flexor surface of the forearm from leaning on it for some time across the sharp edge of a table or desk.

Cervical rib.—An extra rib growing from the transverse process of the last cervical vertebra, may damage the lowest root, which enters the plexus. Consequently the patient suffers pain down the inside of the arm, and anæsthesia of the forearm, and there is wasting of the muscles supplied by the first dorsal root, that is to say the intrinsic muscles of the hand, the interossei and hypothenar muscles usually rather more than the thenar. I have here a case of damage as the result of a splint and the pressure of a bandage. Here is a boy who broke his arm a little while ago and was put up in a splint and bandage, and very quickly afterwards his hand was found in this condition. You can see the tremendous amount of contraction which is present in these muscles. I cannot get his fingers out straight. If I flex the wrist down as much as I can, I can get the fingers straight, but if I try to keep them straight as I extend the wrist I cannot manage it. That happened within a few days of the application of the splint and bandage, and therefore it is not a late paralytic contracture as a result of neuritis or any other nerve lesion. As it was described by Volkmann it is known as *Volkmann's contracture*. Another name for the disease is ischæmic myositis. The result may be brought about without any splint at all: simply by tight bandaging, by which the blood is prevented from reaching the skin and

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muscles, which therefore suffer in their nutrition. A sort of rigor mortis of the muscles ensues, and they become fibrous and extremely contracted. But the muscles do not become degenerated. If you investigate them electrically you find there is no reaction of degeneration as a rule; the muscles will react to faradism, though they will probably require a slightly stronger current to make them respond. Though there is this extreme contraction in this boy, the muscles react fairly well to faradism. However, you might expect that if muscles can suffer from tight bandaging then the nervous structures which run through them might suffer, and occasionally one does find the reaction of degeneration present in those muscles which are damaged. Perhaps more often you will find the reaction of degeneration in the muscles of the periphery of the limb. In this case, although the particular muscles which are damaged do not show the reaction of degeneration, the intrinsic muscles of the hand do show reaction of degeneration—that is to say, the median and ulnar where they pass through the area of damaged muscle have suffered also from the pressure. I have seen several cases of this kind, in which the most marked reaction of degeneration was in the periphery of the limb, not in the muscles primarily damaged by the splint. The same form of neuritis may result from the application of a tourniquet. The treatment of this condition is a very tedious and difficult one. You want to rectify the condition of these muscles and tendons, and the best way which has yet been described, I think, is constant and persistent passive movement and massage. It requires a great deal of patience, it means daily rubbing, for fifteen or eighteen months, or possibly longer. But most of these cases will get well if they are treated long enough. Another method has been tried by operation. The two which are most commonly performed are to either cut down and take out a piece of the radius and ulna and shorten the forearm, bringing the bones down to the length of the tendons, or to cut down from the flexor aspect and lengthen all the tendons. That is a very ticklish operation because you have to take each tendon in turn and split it, and divide the halves at top and bottom separately, slip them along and suture up again. It is a difficult operation and not a satisfactory one according to the surgeons, and I have not seen a very good result from it myself. Most surgeons are not very keen about doing that operation, and the bone-shortening operation is a more simple one, and I think it is more useful in its results.

The next group of nerve injuries which I want to mention is that due to *chill*. The commonest nerve in the body to suffer paralysis as a result of chill is, of course, the facial. But still the nerves of the upper extremity sometimes suffer in this way, that is to say isolated nerves, and sometimes the plexus. I have seen the fifth cervical suffer from nerve palsy as the result of exposure to cold; there was acute pain down the arm followed by typical Erb's paralysis. I have seen the serratus magnus suffer, and I have seen the affection confined to the deltoid and the spinati. I have seen the trapezius suffer, and several such cases, curiously enough, happened after operation, though I cannot believe it was due to this cause. I explain the occurrence in this way in hospital: I believe it is the draught from the windows which are commonly at the back of the patients' beds, and the cold draught struck the shoulder in that region. In one case it came on two weeks after the operation, and in another case ten days, and so on. So we have to remember that nerves may suffer as the result of chill.

The last group I want to speak of is the *toxæmic*. Here we have several causes. One of the most potent, I think, is influenza. The next patient I have to show you is suffering from general brachial neuritis as the result of influenza. One sees brachial neuritis occasionally with a pleurisy, perhaps of influenzal origin, and I have seen it precede the development of severe tuberculosis. Other toxins, such as lead, and diabetes, and alcohol may occasionally strike isolated nerves in one upper extremity, and you know that these three poisons may cause general, multiple, or peripheral neuritis of both upper and both lower extremities, particularly the lower ones. But symptoms of neuritis may affect one nerve, and of the three alcohol is the commonest, and musculo-spiral palsy as the result of alcohol is not rare. I cannot explain it. One knows by experience that it is fairly frequent in alcoholic subjects to get musculo-spiral palsy, in which you cannot trace any actual pressure. One cross-questions such people about sleeping with the arm over the edge of a chair, but that can be eliminated because the condition may come on while they

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are awake, in the day-time. One tries to explain it by saying that these people, being soaked with alcohol, are on the brink of general peripheral neuritis, and that a very slight cause, an amount of pressure which might be entirely neglected in the ordinary person, might precipitate a neuritis, whereas in the ordinary individual nothing would happen. *Gout and rheumatism* are also two potent causes of neuritis. This woman had, last February, a severe attack of influenza. She was laid up in bed with it, and afterwards she noticed intense pain commencing in the arm. That swelled up and the pain was agonising. After the acute attack passed away she found the fingers became rigid, and useless and shiny. You can see how shiny the first two fingers are, and not only that, but the joints are distorted. The terminal phalanx is flexed and I cannot extend it, and the interphalangeal joint is hyper-extended. Notice how wasted the finger-pad of the index finger is. Wasting of the finger-pads is very common in neuritis, and it is a very important point in the diagnosis of neuritis in the periphery of a limb. More than that, she has anæsthesia, which I will not demonstrate now, and some pain. These fingers are stiff from the presence of peri-articular adhesions. There is perhaps no severe arthritis, no destruction of the cartilage or erosion of bone, nothing approaching severe rheumatoid arthritis or osteo-arthritis, and yet the joints are extremely fixed by peri-articular adhesions. That is a common result of severe neuritis, and it is a point not sufficiently remembered, because such a condition as this might be easily mistaken for rheumatism or rheumatoid arthritis. In brachial neuritis the pain usually starts somewhere about the shoulder, down by the side of the scapula, and the pain radiates up into the neck and down the arm; the whole limb is intensely tender and the hand is often swollen. Sometimes the pain involves even the chest, and there has been even cardiac irregularity and heart-burn as the result of brachial neuritis. And this pain, you can easily see, may lead to your mistaking a brachial neuritis for angina pectoris. But it is more commonly mistaken for rheumatism than for anything else. Sometimes it is mistaken for neuralgia. The character of the neuralgic pain will be very much like that of brachial neuritis, so you must distinguish it by other features than by the pain and that is by looking for signs of nerve injury. In true neuralgia, or in a case of referred pain, you will find no signs of nerve injury; there will be no glossy skin, no muscular wasting, no reaction of degeneration in the muscles, and no wasting of finger-pads, neither will there be any peri-articular adhesions. A neuralgia, then, can be easily distinguished in most cases from brachial neuritis or any other form of neuritis. But rheumatism may simulate it more closely. Rheumatoid arthritis may distort the joint in a condition very similar to this, and rheumatoid arthritis may cause wasting of muscles. Wasting of muscles is a common result of the joint lesion, and so the muscles which move the joint become wasted, and on that account this condition is called arthritic atrophy. Arthritic atrophy is familiar to us all in the shoulder and in the hip in chronic rheumatism or tuberculosis of the joints. In the latter the glutei are especially wasted. There is very marked wasting of the muscles which move the joint, and it is frequently miscalled the atrophy of disuse. Now, disuse does not cause atrophy like that, and I have brought a patient to show you this very point. The fact that there may be no atrophy in a muscle which has not been used will be a distinct point in the diagnosis. Adhesions in a joint are, as I have said, very common as the result of neuritis. On the other hand you may get a primary lesion in a joint from rheumatism or rheumatoid arthritis, and there may be wasting of muscle secondarily, so it may be easily mistaken for brachial neuritis. There will, however, be no glossy skin, no anæsthesia, and no reaction of degeneration, but the pain and the joint adhesions and the wasting of muscles may simulate the results of neuritis very closely.

I want to speak of the treatment of such a condition as brachial neuritis. The pain is intense and the limb must therefore be kept at rest, with the forearm flexed, and the arm must be supported and wrapped in thick layers of cotton-wool so as to keep it warm. Hot applications in addition to the warm wool sometimes do good; fomentations, with liniment of belladonna and aconite and chloroform, or opium applications. Radiant heat applications sometimes relieve, and sometimes electrical applications may relieve, very notably by giving the constant current, with a large sponge electrode above the clavicle and the forearm in water. The sponge should be soaked in solution

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of salicylate of soda, and attached to the negative pole, using a current of 10 to 15 ma. In this way you may relieve the pain very much, just as you may relieve sciatica, which is a similar condition to that type of brachial neuritis. It is simply a peri-neuritis attacking the lumbo-sacral plexus in the one case and the brachial plexus in the other. Later, when the pain has gone off, the patient may bear gentle massage and perhaps some stimulant form of treatment and movement of the joint. As soon as the pain has gone there should be massage and passive movements combined, because of the danger of fixation of the joints by peri-articular adhesions. The shoulder-joint is one of the most liable to be fixed by adhesions. I have here another patient, to illustrate the absence of muscular wasting being a guide to diagnosis. She has a stiff shoulder, which she has had for some months. She has had a great deal of pain and yet there has been no wasting of the muscles. The shoulder is absolutely rigid, you cannot move it; you can only get a to-and-fro movement out of it, but there is no true movement of the shoulder-joint—it is all movement of the scapula. The humerus is fixed to the scapula by something; there is nothing to be felt in the way of grating or creaking due to adhesions. The question is, is this fixation due to adhesions or is it due to muscular spasm, or to both? She had been all round the departments of the hospital before I saw her at the nervous diseases department. The first thing which struck me was that there is no wasting of the deltoid or spinati. If the stiffness were due to a severe lesion in the shoulder-joint I should expect to find atrophy of the deltoid and spinati muscles. You will see that these muscles are quite good, as good on the right side as on the left, and electrically they react as strongly on each side. Yet if I try to move the shoulder you will find there is no true movement in the shoulder-joint at all. I cannot rotate it, and I cannot feel creaking of adhesions of any kind. Still, there might be adhesions there which are fixing it. One could not tell what was the matter without an anæsthetic. The fact of there being no wasting suggested that she had no severe lesion of the joint, so I thought we had better put her under an anæsthetic. For a long time during the induction of anæsthesia it was quite rigid, but when she got deeply under and became stertorous, the shoulder became limp and could be moved in any direction, with the exception that in rotating it at first it was held for a moment; I felt a little creaking and the yielding of something as if there was a weak adhesion of the shoulder-joint, which required scarcely any force at all to break it down. No doubt there was some primary rheumatic lesion. But there was no severe lesion of the joint, consequently there was no atrophy of muscles. Yet that joint had been as much disused as any shoulder could have been, and if muscles did atrophy from disuse this deltoid and these spinati muscles must have atrophied. Why do muscles atrophy in these joint lesions? for we know they do waste, sometimes severely. It is a reflex from the joint lesion, and Sherrington, by experiments, has found that it is a reflex atrophy, which is due to afferent stimuli passing up to the spinal cord along the nerves which come from the neighbourhood of the joints, that is to say, that all the nerves which supply the joint carry impulses to the spinal cord, and when a joint is damaged these impulses come from the joint up to the spinal cord, and reflexly alter the nutrition of the anterior horn cells supplying the muscles that move the joint. Owing to that alteration in the nutrition of these cells the muscles may waste rapidly, and so in severe rheumatoid arthritis there may be extreme atrophy, or in the tubercular shoulder or hip there may be extreme atrophy of the muscles moving those joints. Electrically you will find no reaction of degeneration in the muscles, and there is no neuritis of the motor nerves. The stimuli have gone up those afferent nerves, which Sherrington proved by cutting all the afferent nerves from the joint. He produced lesions of the joint and saw atrophy of muscles follow. He then produced a perfectly similar lesion and at the same time cut all the posterior roots which could convey afferent impulses from that region, and he then found that no atrophy of the muscles followed the lesion of the joint, proving that the wasting of the muscles is due to stimuli passing upwards to the posterior roots from the nerves which supply the joint. Therefore in rheumatoid arthritis, or any other rheumatic lesions of any joints, the muscles do not waste as the result of disuse, but the wasting is a reflex effect which is no doubt protective, in order to prevent movements of the joint. What is the moral of that as applied to this shoulder? There was no wasting, showing that there was no lesion of the joint of any seriousness. The condition of the joint under an anæsthetic proved that too. The fixation is due to muscular spasm of a functional nature; it has been overdone in the effort to keep the joint fixed. There is no doubt some slight rheumatic lesion there, but nothing of any seriousness, and the condition of the joint under an anæsthetic proves that. Therefore the determination of the fact that there is no atrophy of the muscle is a great help in the diagnosis, and also in the treatment. When this joint has been moved freely, and she has been encouraged to move it higher and higher day by day, there is no doubt that we shall get the joint perfectly free again.

March 2nd, 1908.

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THE RÔLE OF THE PNEUMOCOCCUS IN OCULAR INFECTIONS.

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MODERN bacteriological researches have taught us that the *Pneumococcus lanceolatus* plays no small part in the causation of several diseases of the eye and of its appendages. For example, it has been found in inflammations of the lacrymal gland and sac, of Tenon's capsule, of the connective tissue of the orbit, of the eyelids, of the conjunctiva, and of the cornea, and, lastly, in metastatic forms of panophthalmitis.

Before describing some of these infections more particularly, it should be said that the pneumococcus has been demonstrated in the normal conjunctival sac, where it appears to be capable of leading a purely saprophytic existence. Gasparrini, who was the first to point out this fact, met with the organism in no less than 80 per cent. of his cases ('Annali di Ottalmologia,' xxii, 6, p. 488). His figures, however, have been challenged by other writers, especially by Axenfeld and Oertzen ('Die Bakteriologie in der Augenheilkunde,' Jena, 1907, p. 38), who examined forty-nine healthy eyes, and twice only obtained cultures of the pneumococcus on glycerine-agar. The micro-organisms thus found by them exhibited a low degree of vitality, and were scarcely pathogenic as regards animals. Again, A. Lawson ('Transactions of the Jenner Institute of Preventive Medicine,' 1899, p. 56) examined twenty normal conjunctival sacs and found Fraenkel's organism twice only, in both instances in the same patient. The growth died out before Lawson had an opportunity of testing its virulence.

From what has been said we may conclude that the pneumococcus, probably in an attenuated form, can be found only now and then in the healthy conjunctival sac.

That the pneumococcus is pathogenic as regards the eyes of certain animals, as the rabbit, has been proved by the experiments of Guaita, Gasparrini, Hiroda, Römer, de Lapersonne, and others. Broadly speaking, inoculation of the conjunctival sac of those animals produced an inflammation of the conjunctiva; of the cornea, a purulent keratitis, with or without pus in the anterior chamber; and of the vitreous humour, a panophthalmitis.

Inoculations of the human conjunctiva have been made by Hauenschild, Gifford, and Veasey. Hauenschild ('Zeitschrift f. Augenheilkunde,' 1900), set up conjunctivitis in his own eye by inoculating the pneumococcus. H. Gifford ('Archives of Ophthalmology,' vol. xxv, 1896, p. 314) repeated Hauenschild's experiences with pus from a case of pneumococcal conjunctivitis upon his own eye, as well as upon that of an assistant. In addition to this, he obtained a positive result in two patients to whose conjunctival sac he applied an anaërobic culture of the third generation. He gave reasons for believing that in some cases one attack of pneumococcus conjunctivitis exercised a temporary immunising influence upon the affected conjunctiva. C. A. Veasey ('Archives of Ophthalmology,' vol. xxviii, 1899, p. 301), obtained a positive result by inoculating a patient's conjunctiva with discharge taken from a case of severe pneumococcus ophthalmia. Successful results also followed the inoculation of (*a*) an anaërobic culture of the second generation, and (*b*) a pure aërobic culture.

One of the most interesting experiences was that related by Hallé ('Annals d'oculistique,' t. 123, 1900, p. 200). A medical man inoculated his own eye during the performance of a thoracentesis for the relief of a pneumococcal pleurisy, and seven days later (*sic*) developed a pneumococcal inflammation of the conjunctiva. Hallé also mentioned another case where conjunctivitis followed three days after the entrance into an eye of the saliva and nasal secretion from a child affected with pneumococcal conjunctivitis.

Among the numerous micro-organisms found in the discharge from a lacrymal sac chronically inflamed on account of some obstruction in the naso-lacrymal duct, the so-called "dacryocystitis," none is of more importance than the pneumococcus, as shown by the researches of Gasparrini, Uhthoff, and Axenfeld. Pneumococci from such a case are apt to inoculate any trivial abrasion of the cornea that may chance to be present, and may thereby give rise to that destructive affection of the eye known in this country as "serpiginous ulcer of the cornea" or "hypopyon-keratitis," and

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in Germany as "ulcus serpens." It is the knowledge of this fact that causes the ophthalmic surgeon to lay such stress on the cure of catarrhal affections of the lacrymal passages.

As to the relative frequency of the organism in dacryocystitis, Cuénod found it to be present eight times (80 per cent.) in ten cases; Gerstenberger, four times (33 per cent.) in twelve cases; and Brons, sixteen times (53 per cent.) in thirty cases. It would be a safe generalisation to say that the secretion of an inflamed lacrymal sac contains pneumococci, pure or mixed with other micro-organisms, in one-half of all cases.

There is an interesting condition not uncommon* in babies, where owing to delayed developmental processes, the naso-lacrymal duct, instead of being patent at birth, as it should be, is more or less closed by mucous folds or filled with inspissated secretion from liquefaction of the epithelial cells, which in fœtal life form the rudiment of the lacrymo-nasal duct. Should infection occur, as it is apt to do either before or shortly after birth, we have a kind of dacryocystitis produced, or in rarer instances, an actual lacrymal abscess. In this condition the pneumococcus is often present. Thus, in twenty of my own cases, examined by means of smears and cultures, and confirmed in some instances by animal inoculation, pneumococci were found in thirteen—that is to say, in pure culture three times, and associated with *Staphylococcus albus* and *aureus*, streptococcus, *Bacillus coli*, *Pneumo-bacillus*, *Cocco-bacillus fœtidus ozænæ*, *Bacillus buccalis max.*, *Bacillus subtilis*, *Bacillus xerosis*, and a strepto-bacterium in the remaining cases, ten in number.

The pneumococcus, as already stated, is pathogenic as regards the human conjunctiva, in which it is capable of producing varying degrees of inflammation. Pneumococcal conjunctivitis may be considered as it affects: (1) newly-born babies, and (2) older subjects.

(1) Ophthalmia neonatorum is caused in about two thirds of all cases by the gonococcus, derived directly or indirectly from the maternal passages. Of the remaining cases no inconsiderable proportion are associated with the pneumococcus. In round numbers, 10 per cent. of all the cases are due to the pneumococcus. We may conclude that next to the gonococcus the pneumococcus is the commonest micro-organism associated with ophthalmia neonatorum. For the first observation of this kind we are indebted to Victor Morax ('Thèse de Paris,' 1894), who described such a case in a baby, æt. 8 days. Since then cases have been reported by many writers, including Parinaud (1894), Gasparrini (1894), Axenfeld (1896), Schuhl (1897), Gonin (1899), Groenouw (1899), v. Ammon (1900), Alt (1901), Brewerton (1903), Pollock (1905), Augé (1906), Wharton (1907), and myself. The cases, as a rule, are mild, seldom entail corneal damage, and present the peculiarity of improving more or less suddenly after lasting four or five days. It has been suggested by Cramer ('Centralbl. f. Gynäk.,' October 14th, 1899) that in some instances the eyes of the baby may become infected by the saliva of the mother, nurse, or friends. While not denying this possibility, for which, indeed, there is some clinical evidence (Wharton), it is more probable, I suggest, that the organism is commonly derived from the mother's genito-urinary organs.

The pneumococcus in rare instances may become generalised. For example, I have seen two cases of pneumococcus ophthalmia neonatorum associated in the second week of the disease with a subacute inflammation of the knee-joint. The arthritis, which was at no time severe, subsided practically without treatment. The cases are clearly analogous to those of gonorrhœal arthritis, first described by Mr. R. Clement Lucas ('British Medical Journal,' February 28th, July 2nd, and October 10th, 1885) and by Professor Widmark ('Beiträge zur Ophthalmologie,' 1891, p. 95). I have published elsewhere ('Ophthalmia neonatorum,' London, 1907, p. 143) a case of mild septicæmia following ophthalmia neonatorum, where there were some grounds for thinking that the pneumococcus was the infecting agent.

(2) In older subjects pneumococcal conjunctivitis may appear under various clinical guises. Although, in my experience, not common in London, yet it seems to be common enough in some other parts of the world. Gifford states that it is the most frequent form of conjunctivitis in the district of Omaha, U.S.A., and Veasey that it is common in Philadelphia. From certain parts of Germany similar evidence is to hand. Few of the cases

* Among 1538 out-patients seen in the Eye Department of the North-Eastern Hospital for Children, London, no fewer than 27, or 1·75 per cent., were affected.

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I have personally seen could be recognised as pneumococcal except with the aid of the microscope. Still, there are one or two signs that, when present, and especially when grouped, enable us to diagnose the ailment with more or less certainty. These include the co-existence of coryza, the presence of a pellicle of fibrin on the surface of the palpebral conjunctiva, or of small multiple ecchymoses into the ocular conjunctiva, and, lastly, of a fleeting rose-coloured puffiness of the free edge of the eyelids.

This form of ophthalmia shows a special tendency to affect children, and when grown-up people are attacked its symptoms (as pointed out by Axenfeld) are usually of a more severe type. It is generally bilateral. de Schweinitz's case ('Annals of Ophthalmology,' 1906, p. 191), where an acute pneumococcal conjunctivitis was followed by a large bulla (5 mm. by 3 mm.) on the centre of the cornea, must be unique.

Last, but not least, we pass forward to consider briefly pneumococcal infections of the cornea, the practical importance of which it would be difficult to over-estimate.

In 1889 Widmark (ref. in 'Archives d'Ophtalmologie,' 1889, July, p. 371) mentioned a case of serpiginous ulceration of the cornea in which he had found an organism answering to the description of the pneumococcus, and in 1893 Gasparrini ('Annali di Ottalmologia,' 1893) announced that he had obtained cultures of the same organism from numerous cases of hypopyon-keratitis. The pioneer work of these earlier observers (there were others whose names need not be specified) was confirmed and extended in the following year, when Uhthoff and Axenfeld published their well-known work, in which they stated that the ulcus corneæ serpens, described twenty-four years before as a clinical entity by Saemisch ('Das Ulcus Corneæ Serpens und seine Therapie,' 1870), was generally due to the action of the pneumococcus. In 1896 the same writers ('Archiv f. Ophthalmologie,' xlii, 1, 1896) reported upon fifty cases of ulcus serpens, in which pneumococci had been found in 33, or 66 per cent. In a third communication Uhthoff and Axenfeld ('Archiv f. Ophthalmologie,' Bd. 44, 1897) reported a further series of sixty-eight cases of serpiginous ulcer, a number that included some that were atypical, although associated with the pneumococcus. Doetsch ('Die Ophthalmologische Klinik,' October 5th, 1900), three years later, found pneumococci forty-one times among sixty-three cases of hypopyon-keratitis met with in the Jena Eye Klinik during the years 1898 and 1899. Salvaneschi ('Annali di Ottalmologia,' vol. xxxv, 1906, fasc. 1–2) in thirty-four cases of hypopyon-keratits found the pneumococcus thirty to twenty-six times in pure culture and four times mixed with other micro-organisms. He believed that its virulence was enhanced under the former circumstances.

Römer declares that in 95 per cent. of all cases of serpiginous ulcer of the cornea the pneumococcus may be demonstrated. For that matter, it may be pointed out that his method of treating ulcus serpens by the intra-muscular injection of dead cultures of the pneumococcus implies that all cases of the disease are due to that micro-organism. Indeed, it is obvious that the absolute frequency with which the pneumococcus is found in ulcus serpens will differ according to the methods of investigation employed. In order to obtain the best results it is necessary: (1) to collect material, not from the floor of the corneal ulcer, where pneumococci are scanty and more or less inert, but from beneath its advancing and yellowish-white edge, where the organism exists in a state of activity; and (2) to employ nutritive media particularly favourable to the growth of the pneumococcus, such as that used by Römer, which consists of bouillon to which one third part of sterile rabbit's serum has been added.

Thanks largely to the experimental researches of Professor Leber we are now well acquainted with the pathology of hypopyon-keratitis. The pneumococcus, derived either from a diseased lacrymal sac or conjunctiva, or from a foreign body itself, or indirectly from the saliva * or nasal secretion, gets access to the cornea through some abrasion † which is often so trivial as to excite neither the attention

* G. Hotta ('Klin. Monatsbl. f. Augenheilkunde,' September, 1905) injured the cornea of rabbits in several ways and then inoculated the wounds with human saliva. Positive results, as shown by iritis and hypopyon, obtained whenever the cornea was "pocketed." Pneumococci found in 36 per cent. of these cases.

† Uhthoff and Axenfeld ('Archiv f. Ophthalmologie,' xlii, i, p. 1) nevertheless failed to find the pneumococcus in objects by means of which corneal abrasions are commonly inflicted, as morsels of stone, twigs, and ears of wheat.

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nor the solicitude of the patient. The breach of surface is essential. We know that pneumococci are unable to attack the intact epithelium of the cornea. Once in the *substantia propria*, however, the organism spreads out laterally between the lamellæ and proceeds to elaborate its toxines. Now, although Descemet's membrane is germ-proof, yet it opposes no barrier to the diffusion of toxines. The latter, accordingly, are enabled to pass through the membrane and in that way to reach the aqueous humour and the structures, as the iris, in contact with the humour. The toxines exercise a noxious influence upon the blood-vessels of the iris, ciliary body, and the parts about Fontana's spaces, whereby leucocytes are caused to migrate into the anterior chamber, where they become visible under the guise of a larger or smaller collection of pus (hypopyon). This process explains the fact (which is disputed by nobody who has undertaken the necessary investigations), namely, that the pus in the anterior chamber in cases of hypopyon-keratitis is free from bacteria. The only exception to this rule is to be found in cases where the cornea has already perforated. The older observers had noticed that in some cases a filament of pus appeared to connect the corneal infiltration, on the one hand, with the hypopyon, on the other. This appearance they interpreted as meaning that the pus in the chamber was due to a backward extension of the suppurative process going on in the cornea. We now recognise that in point of fact it is owing to a process of chemotaxis.

Typical ulcus serpens, as outlined above, occurs only in middle-aged or elderly persons, usually of the labouring classes, who are naturally more exposed to injuries of the eye than others better placed in the social scale, and who, besides, are apt to attach little importance to a watery or discharging eye. Pneumococcal infection of the cornea is commoner during the warm summer months than at other seasons of the year. In a large number of the patients, estimated at from 40 per cent. (Angus MacNab) to 75 per cent. (Otto Haab), there are symptoms of lacrymal obstruction; and, as stated before, pneumococci are present in about one-half the cases.

The hypopyon-keratitis of children, as a rule, is a much milder affection, yields to simple treatment, and seldom entails the loss of an eye. It may, indeed, be doubted if a typical ulcus serpens has ever been observed in childhood (Axenfeld). The starting point of hypopyon-keratitis in children is nearly always an ulcerated corneal phlyctenule. The breach becomes inoculated with pyogenic organisms, and hypopyon is brought about in much the same way as in adults. At the same time, in my experience, the pneumococcus is often present. Of seven of my own cases the pneumococcus was identified in four instances—twice in pure culture and twice mixed with staphylococcus and the xerosis bacillus. In the remaining three cases the diplobacillus of Morax-Axenfeld was present, either pure or with the xerosis bacillus as the only contamination.

The last corneal condition in which pneumococci are not uncommon is known by the name of "keratomalacia"—that is to say, a kind of gangrene of the cornea apt to supervene in young children much enfeebled by wasting disease, as tuberculosis, syphilis, ileo-colitis, and so forth. The pneumococcus has been found in these cases by others besides myself, as v. Hippel, Knaebel, and Doetsch. At the same time there is no reason for believing it to be the cause of the disease, which is essentially one due to the lowered vital resistance of the body in general and of the cornea in particular. The pneumococcus may not be present. Other organisms, as staphylococci, streptococci, *Bacillus coli*, may be found in the ulcerated cornea. It seems likely, therefore, that in the special circumstances under which keratomalacia occurs, almost any pyogenic microbe may suffice to produce the corneal mischief.

March 2nd, 1908.

We have received from the Caxton Publishing Company, of Clun House, Surrey Street, London, the first section of the 'Edinburgh Stereoscopic Atlas of Obstetrics,' edited by G. F. Barbour Simpson, M.D., F.R.C.P.E., F.R.C.S.E., and Edward Burnet, B.A., M.B., Ch.B. This part of the publication contains twenty-five stereograms of the normal and abnormal pelvis. The Editors have done most excellent work in producing this section, and it must be gratifying to them to know that this latest evidence of the forward movement in medical education will most assuredly receive the heartiest welcome from all concerned. In addition to the full description which is attached to each stereogram, the main features are specially indicated above the photograph, so that while the student studies it he can at once recognise the points to which his attention should be particularly directed.

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THE CLINICAL JOURNAL,

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LECTURES ON CHRONIC INTESTINAL OBSTRUCTION.

By H. L. BARNARD, M.S.,

Surgeon (with charge of out-patients) to London Hospital.

LECTURE I.

GENTLEMEN,—In these lectures I propose to consider those conditions which produce the collection of symptoms known as chronic intestinal obstruction. These symptoms are the same as those which are found in cases of acute intestinal obstruction, but they differ very much in degree.

Symptoms.—The first of these is *pain.* It is gradual in its onset and it intermits for long intervals, during which the patient is entirely free from suffering. It is of a colicky, peristaltic character, and is associated with the rumbling of wind within the intestines. *Vomiting* is late in its onset. It only occurs at long intervals, and can frequently be avoided entirely by an abstinence from solid food, so that the patient very frequently tells us that he has not been sick for three or four days, but only because he has not eaten anything. The *collapse* is slight in degree and comes on very late in the condition, so that it is not at all well marked until the chronic intestinal obstruction has passed into its final acute stage, and the patient is poisoned by the foul fermenting contents of his intestines. The *temperature* is for the most part normal, whereas in acute intestinal obstruction you will remember it is, as a rule, subnormal. The extremities are not cold in the chronic condition, nor does the patient suffer from cramps in the calves of his legs.

The *constipation* is incomplete and the patient passes motions which, when the condition is due to a carcinomatous stricture, are often of small

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calibre, and when to chronic fæcal accumulation are formed of scybalous masses. *Blood and slime* are found in the motions when ulceration is present in the colon, whether that ulceration be malignant or simple. In many cases of chronic obstruction constipation alternates with attacks of diarrhœa, due to the enteritis produced by the stagnating contents of the bowel above the obstruction.

An important sign in carcinoma of the colon is the onset, without any obvious cause in a man beyond middle life, of *morning diarrhœa*. Such a patient will develop the habit of emptying his bowels of a fluid motion directly he arises from his bed in the morning and before he has been able to dress himself. This is extremely suggestive, and a carcinoma will nearly always be found on examination of the sigmoid flexure or the rectum. I mentioned this sign to you in my last lecture and one of my audience sent to my Out-patients a case exhibiting this sign. On examination I found a large ulcerating carcinoma high up in the rectum and adherent to the bladder in front. As the growth could not be removed and was not obstructing the bowel, but was of the ulcerating variety, I recommended that colotomy should be postponed as long as possible.

The abdomen in chronic intestinal obstruction is nearly always distended, for the causes of this condition act upon the great gut, and it is rare for great distension to be due to small intestine. *Contracting coils of gut* may be seen in nearly all cases. To observe this sign it is well to darken the room and to allow beams of light to strike across the abdomen so that the shadows of the irregularities in the abdominal wall are well revealed. It will then be seen that worm-like movements are taking place at intervals in the distended coils of intestine. The presence of this sign implies that there is definite organic obstruction to the intestine and that this obstruction has been present for at least two or three months, for it requires this lapse of time to produce the hypertrophy and distension of the intestine combined with the wasting of the abdominal wall which produce this important sign. I have been very much struck in the last few months by the importance of the history of *rumbling of wind about the abdomen* in the diagnosis of chronic obstruction, and when inquiring about this symptom I always endeavour to discover whether it is of recent origin. If a patient of fifty years of age, who previously suffered from no abdominal disturbance or dyspepsia, has, during the last six months, experienced considerable annoyance from the rumbling of wind about his abdomen associated with slight colicky pain, it is highly probable that some cause of progressive obstruction of the intestine is present, and this is most likely to be carcinoma of the large intestine. I have found that the obstruction can usually be located in the colon by inquiring on which side the flatulence is experienced and at what point it stops and severe colicky pains are felt. For instance, if the stricture be in the sigmoid the patient tells us that the wind rumbles across the upper part of the abdomen and down the left side to the region of the left groin, when a large lump rises up which is frequently described as being as large as an orange or a cocoanut, and he then experiences sharp, colicky pains until he has rubbed away the mass with his hands. This is, of course, his description of a peristaltic wave sweeping along the transverse and down the descending colon, and endeavouring to force their accumulated contents through a narrow stricture in the sigmoid. When the stricture is in the splenic flexure the rumbling of wind will be on the right side of the abdomen and across its upper part, and the pain will be in the region of the spleen. In a similar way when the hepatic flexure is the site of carcinoma the flatulence will be confined to the right and the result of spasmodic contraction will be pain in the right hypochondriac region. In a few instances, moreover, the application of a stethoscope to the suspected spot will enable us to hear the mixed gaseous and fluid contents being squeezed through the stricture and producing a characteristic sound.

These symptoms will enable you to recognise that you have to deal with a case of definite organic but chronic intestinal obstruction, though it will be necessary to go a little further and to determine which of five conditions is producing this obstruction, for these conditions require not only very different treatment but they are of very diverse gravity, both at the time and as to the prognosis. These conditions may be enumerated as follows:

(1) Fæcal accumulation.
(2) Carcinoma of the colon.
(3) Pressure on the intestine from without.
(4) Chronic intussusception.
(5) Simple strictures.

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Chronic Constipation, Fæcal Accumulation and Coprostasis.

Of the 669 cases of intestinal obstruction which I have collected from the London Hospital records, 1893–1905, 69 were due to fæcal impaction, and of these 32 occurred in men and 37 in women. It is therefore manifest that this is a common cause of obstruction and that it is nearly as common in men as in women.

Ætiology.—Fæcal accumulation is a disease of the colon and constipation is due to disordered function in that organ, but when we inquire definitely, What are the normal functions of the colon? we find that very little is known for certain of that organ. Everard Home showed that a large amount of absorption took place in its upper part and that fat was absorbed there in ducks. Cellulose is apparently digested in the cæcum of the herbivora by the action of the intestinal micro-organisms, and very probably the same occurs in man. W. B. Cannon, by mixing bismuth with the food and in enemata administered to cats, was able to observe the movements of the colon by means of X rays. He found that the upper part of the colon underwent active anti-peristaltic movements during digestion and forced the contents back into the cæcum, churning them up continuously. Arthur Keith has demonstrated the sphincter action of the ileo-cæcal valve and has suggested that the acrid corrosive contents of the ileum are injected into the cæcum in repeated doses, and there are neutralised and their ferments destroyed. This view is confirmed by a comparison of cæcotomy openings, which are nearly always eczematous and corroded, with those lower down the colon, and Sir William Macewen was led to similar conclusions by his observation on a man whose cæcum was laid bare by an explosion. He also suggested that the appendix with its profuse lymphoid tissue may prepare and maintain an attenuated culture of the *Bacillus coli communis* wherewith to inoculate the chyme as it enters the cæcum.

The descending colon, the sigmoid flexure and rectum are concerned in extracting water from the fæces and storing them until a convenient time for their evacuation.

Solid fæcal masses should not normally be present above the sigmoid flexure; they should collect in that loop, and when propelled into the rectum the desire to defæcate should at once be excited.

From the above short review of what is known of functions of the colon we may conclude that in primitive man it was evolved to digest coarse food, consisting mainly of raw fruit and vegetables. In such food the starch granules are often enclosed in cellulose coverings, and these were removed in the cæcum by micro-organisms. We may further surmise that primitive man was forced to active exercise in hunting for his food, and when he was successful ate it with relish and with hunger.

The modern town dweller is provided with a highly nitrogenous diet from which raw vegetables are almost entirely excluded. His food is ground fine and cooked so that the cellulose casings of the starch granules are burst by imbibition. He swallows his food in haste and often without appetite or attention. He no longer is compelled to regular exercise in the daily search for nourishment, so that his lower colon is not stimulated by regular and vigorous contractions of his abdominal wall to expel its contents. Even milk, the most digestible of foods, is partially cooked and deprived of certain of its constituents, whilst the apostle of Pasteurisation of milk in Germany is reported to be coming to London to institute a campaign against the natural diet of infancy. It is really not surprising that under such a change of circumstances the colon should become inert, relaxed and distended with the decomposing refuse of food.

The causes of habitual constipation may be classified as follows:

(1) *Alimentary constipation* includes those cases in which proteids form the major part of the diet, and vegetables, especially raw fruits, are almost entirely excluded. The digestion of vegetables in the colon leads to the formation of gas and their fermentation to the production of acids, and these stimulate intestinal movements.

Thus Rubner found that carrots passed along the alimentary canal in 4 hours, brown bread in 14 hours, white bread in 17 hours, and animal food in 3–4 days.

(2) *Lack of exercise* and a sedentary life.

(3) *Mental work*, preoccupation and worry.

(4) *Irregularity* in emptying the rectum. The influence of habit is well marked in obtaining a regular evacuation of the bowel. Constipation is particularly prone to occur among people of irre-

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gular habits, and has often dated from a break of regularity such as may arise during journeys or prolonged illnesses.

(5) *Muscular atrophy of the colonic wall.*—Nothnagel describes cases in which the colon was found to be only ·12 mm. to ·25 mm. thick, whereas it should average ·5 to 1·0 mm. In cases of chronic tuberculosis and alcoholism the muscular wall of the colon may undergo fatty degeneration.

(6) *Nervous causes of constipation.*—Constipation is common in the insane, hypochondriacal, neurasthenic and hysterical, and in organic lesions of the brain and spinal cord.

In recurrent melancholia constipation often appears and disappears with each attack. Indican may be present in large quantities in the urine of some cases, and its decrease and disappearance frequently herald recovery.

Constipation is, however, probably rather a consequence than a cause of the mental condition, and can only produce mental symptoms in those who are already strongly predisposed to nervous disease.

(7) *Mechanical causes.*—Virchow has always been quoted as the authority for the mechanical theory of habitual constipation, and there is no doubt that at autopsies in some very advanced cases of fæcal accumulation, adhesions, kinking of the flexures, anchoring of the gut, partial volvulus, shortening or lengthening of the mesentery and abnormal positions of the cæcum, transverse colon, and sigmoid flexure have been found. Even Virchow, however, recognised that these were probably the result of previous stages of constipation, and therefore could not be regarded as primary causes of constipation. Moreover, as Nothnagel says, "In reality anchoring of the intestines by peritoneal adhesions is relatively rare as compared with the extraordinary frequency of habitual constipation."

(8) *Weakness of the striated skeletal muscles.*—Constipation is common in the emaciated and in pregnant women and multiparæ, in whom the abdominal wall acts at a disadvantage. Diastasis of the recti muscles and large herniæ act in a similar way.

(9) *Circumscribed atony and spasm* is a determining cause in some cases, and in these opium and belladonna act as laxatives, as in cases of plumbism.

(10) *Pain* may produce constipation either by a very natural disinclination to defæcation or by inducing spasm of the sphincter ani and inhibition of the detrusor. Such pain is due to piles, fissure, and ulcers of the anus, inflamed ovaries, Fallopian tubes or chronic appendicitis.

(11) *Organic causes* include innocent and malignant strictures, pressure of tumours upon the intestine, foreign bodies, and malformations.

The Morbid Anatomy and Pathology of Coprostasis.

The morbid changes which are found in fæcal accumulation are as follows:

(1) Fæcal tumours.

(2) Lengthening of the mesentery with displacement of the colon.

(3) Shortening of the mesentery and anchoring of the gut.

(4) Stercoral ulceration of the colon and colitis.

(5) Distension and pressure effects.

Of these *stercoral ulceration and colitis* are of the greatest importance. The presence or absence of stercoral ulceration is the main factor in determining whether chronic constipation will affect the health of the patient or not.

Stercoral ulcers are common in the rectum and in the sigmoid flexure, but the cæcum is also a favourite site.

In size they vary from that of a shilling or half-crown to huge ulcerating surfaces surrounding the fæcal tumour. Their extent, depth, and severity bear no relation to the size of the accumulation. Some enormous masses are free from ulcers whilst a small and solitary fæcal tumour may rest on a huge ulcerated area.

The effects of stercoral ulceration are of three kinds, (1) discharge, (2) absorption, (3) perforation and peritonitis.

(1) *Discharge.*—This is serous, sanious, or purulent. It is responsible for the attacks of diarrhœa which punctuate the progress of chronic constipation, and which may terminate in continuous incontinence, a very deceptive symptom.

(2) *Absorption* occurs from the raw surfaces of the ulcers. The result will depend on the virulence of the micro-organisms and toxines which are absorbed. In the most severe cases the patient may suddenly die of *intestinal septicæmia*, or more slowly succumb to *intestinal toxæmia*. In slight cases there is little more than malaise, loss of

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energy, a slight degree of anæmia and some loss of flesh. In others the main effect of absorption is attacks of *fæcal fever*, and this is prone to occur in children. When the nervous system of the patient is of an unstable neurasthenic type the absorption of these toxines may produce a sense of pressure and heaviness in the head, vertigo, somnolence, stupor, hypochondriasis, or even attacks of mania and insanity.

In another curious group of patients innumerable colon bacilli enter the blood by the ulcer and pass out through the kidney, producing a variable degree of irritation of the urinary track. This condition is one form of *bacilluria*, and is greatly benefited by frequent and small doses of calomel.

(3) *Peritonitis and perforation.* — Localised peritonitis, which leads to adhesions around the colon, is a not infrequent result of stercoral ulceration. Perforation may also occur into either the retroperitoneal cellular tissues or the peritoneum.

When the perforation is slow the resulting abscess is localised; when it suddenly occurs general peritonitis is produced. In rare instances where the infection of the ulcer is exceedingly virulent extensive areas of the colon may become gangrenous, and this is particularly liable to occur in the cæcum.

Distension and pressure are also important results of chronic constipation. The thoracic and abdominal viscera may all be pressed upon and disordered in their function.

The symptoms of compression of the heart and lungs are palpitations, a sense of anxiety, breathlessness, and orthopnœa. In a recent case which came under my notice an ordinary attack of pneumonia terminated fatally in consequence of fæcal accumulation complicating it.

The vena cava, portal and spermatic veins may be pressed upon in exaggerated cases of coprostasis, and this may lead to œdema of the legs and scrotum, varicocele, and hæmorrhoids. Pressure may be exerted on the bladder or the uterus and its appendages, and the functions of these organs may be interfered with. Finally sciatica and neuralgia of the crural and other nerves may result from the pressure of fæcal masses.

Symptoms of Fæcal Accumulation.

A moderate degree of constipation is much more common in women than in men, but severe forms producing intestinal obstruction are more evenly distributed between the sexes. Of 69 severe cases at the London Hospital 32 were men and 37 were women.

It must be clearly stated that habitual constipation may be present, and yet the patient may enjoy good health. As a rule, however, the subject of habitual constipation suffers from mild alimentary signs, such as fulness, tension, and discomfort in the abdomen, with flatulence and belching of wind, and from a foul taste in the mouth. The stools of such patients are lumpy and often covered with mucus, and are only evacuated by severe straining or aperients. As the case advances attacks of irritative fever or fæcal fever, due to stercoral ulceration and colitis, punctuate its progress.

At a still more advanced stage the results of absorption appear. The patient has a foul taste in his mouth, loses his appetite, his breath is offensive, and he wastes and weakens. His skin becomes dull and greasy. His conjunctiva, eyelids and axillæ are yellowish in colour, and his sweat of an unwholesome odour.

When the nervous system is unstable he becomes dull and lethargic or irritable and morose, or suffers from profound depression, to recover when the lower bowel is emptied. In rare instances mania, insanity, or melancholia may arise from this cause.

In the advanced stages of constipation *fæcal tumours* are often found in the abdomen. These masses have been mistaken for sarcoma and carcinoma of the peritoneum, mesenteric glands, omentum, stomach, liver, spleen, kidney, ovaries or uterus, or for inflammatory collections or abscesses, when they occur in connection with appendicitis, salpingitis, gastric ulcers, or gallstones. Fæcal masses may, however, be recognised by the following means: They are in the course of the colon. The tumours are peculiarly tender and sore, and are resonant on percussion. In some cases they can be indented by steady pressure with the index-finger. Their surface is rounded and never flat. When steady pressure is made upon a fæcal tumour and the pressure slowly relaxed the mucous membrane may be felt to separate with a sticky sensation (Gersuny's "adhesion symptom"). A large collection of scybala is often found in the rectum. A turpentine

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enema will nearly always bring away hard and old fæcal material, and fragments of food such as pips and currants may be recognised which have not been eaten for days or even weeks, and it frequently is found that a series of enæmata produce increasing large results. This is a very sure sign.

Ultimately fæcal accumulations lead to attacks of colic, and these may pass on to acute distension and paralysis of the intestine, but contracting coils of intestine are seldom seen. These attacks are frequently precipitated by some violent purge which has not acted, and after several such attacks have been relieved by enemata and opium these means at last fail and the patient dies of ileus of the intestine with profound collapse and vomiting.

The Treatment of Fæcal Accumulation.

We will first consider the main lines on which chronic constipation should be treated.

(1) *The diet* should contain plenty of vegetables and raw fruit, which will occupy and stimulate the colon, and proteid matter should be diminished because it leaves little residue and is digested in the upper part of the alimentary canal. Fluids should be freely taken, for constipation is often due to a habit of drinking little; on the other hand, astringent red wines, tea, chocolate, and cocoa should be avoided. A glass of warm water night and morning is often beneficial. The food should be well masticated and the teeth attended to. Any idiosyncrasy of the patient must receive attention, for many foods constipate some people and relax others. This is especially so in the case of milk.

(2) *Muscular exercise* must be insisted upon in the form of walking, horse-riding, cycling, lawn tennis or rowing, fencing, Swedish exercises, or a training in regularity combined with exercise which may be secured at a gymnasium. Excess to the point of fatigue must be carefully avoided, and the treatment must be persevered in for months or years.

(3) *Massage* is adapted for those whose lack of vigour and muscular development do not permit of exercises. It should be carried out by a specially trained person. The object is to stimulate the movements of the colon and not to force the contents along it. When symptoms of stercoral ulceration are present massage must be practised with the greatest possible care.

(4) *Electricity.*—The Faradic current is employed. The object is to stimulate the abdominal and intestinal walls. In "the abdominal method" both poles are well wetted and are then pressed deeply into the abdomen at short intervals. In other methods an insulated metal ball is passed up the rectum and the other pole is applied to the abdomen or the spine.

(5) *The hydropathic treatment* is carried out in special institutions. It consists of frictions, hot and cold douching, sprays, and wet binders.

(6) *Regularity* of habits, but especially in the matter of defæcation, must be insisted upon. The patient must endeavour to defæcate after breakfast every morning even if he is often unsuccessful. He must eat at regular hours and devote due time and attention to the process. Constipation, like insomnia, is in a large degree a bad habit.

(7) *Medicinal treatment.*—As far as may be, drugs should be avoided and the above measures relied on.

Purgatives, such as calomel, castor oil, colocynth, and jalap, should only be used to empty the colon at intervals.

Laxatives, such as cascara, rhubarb, and aloes, may be used to increase the fluid poured into the large intestine.

Moderate doses of saline waters (Marienbad, Carlsbad, Homberg, Kissengen) taken on an empty stomach before breakfast are often effectual in clearing the bowel but their effect is not curative, and the dose tends to constantly increase and the constipation is confirmed. More especially in young girls constipation often has a mental side, and arises from the conspicuous position of the only available water closet.

The Treatment of Fæcal Colic and Obstruction.

As soon as fæcal accumulation has produced colic, distension, and vomiting, all purgatives must be abandoned, for they will only further distend an overloaded colon and flog into action a muscular wall already exhausted. If it is perfectly clear that the obstruction is due to coprostasis, opium may be given in moderate doses to relieve the spasm and pain.

The obstruction itself should be removed from below by repeated enemata assisted by gentle massage. Soap and water will suffice where the mass is soft and recent, but where it is hard and

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the colon is sluggish turpentine may be added in the proportion of from ʒss–ʒij per pint. Brine enemata (1–10) powerfully stimulate the intestinal wall, and castor oil, Epsom salts, alum, and assafœtida have also been added to enemata. In obstinate cases a pint of olive oil or a couple of ounces of glycerine may be introduced high up into the rectum, and after a long interval a turpentine enema will often produce a large result.

The method of lavage may be employed in the case of huge, hard collections of fæces. A gallon or more of normal saline solution or hot water is used at a temperature of 100° F., and is directed in a continuous stream through a byeway tube upon the fæcal mass. The irrigator should not be raised more than three feet and the patient is placed upon the right side with the hips raised. Where a very hard fæcal mass occupies an ulcerated rectum the sphincter should be dilated under an anæsthetic and the collection broken up and washed out.

The Surgical Treatment of Chronic Habitual Constipation.

From the above account of the organic changes which occur in a colon the seat of fæcal accumulations, it is evident that in the latter stages of the disease there is little hope of a return to normal structure and functions, or of the patient regaining even a moderate degree of health and well-being.

It is under these circumstances that surgery has stepped in to relieve a condition which until recently was regarded as entirely in the domain of medicine.

Mr. Mansell Moullin in October, 1900 ('The Medical Society's Transaction,' vol. xxiv, p. 199) was apparently the first to unite the ileum to the lower part of the sigmoid flexure by lateral anastomosis in a case of chronic constipation. His patient was a woman who for seven years had suffered from atony of the colon and obstinate constipation following an attack of acute colitis. The patient was cured of her constipation.

Mr. Arbuthnot Lane was, however, the first to definitely propose to treat chronic constipation by exclusion of the colon from the alimentary canal. The operation is a simple one, the mortality is low, and the immediate benefit is great. Mr. Moullin's patient was still relieved of her constipation after four years when she was last seen.

A serious result however, from exclusion of the colon in many cases is the accumulation in the blind end of inspissated mucus and epithelial *débris* which may form large masses. Mr. Harrison Cripps ('British Medical Journal,' January 20th, 1906, p. 143), referring to more than 600 cases of colotomy, said that in from 20 to 30 per cent. there was trouble from decomposing mucous secretion in the distal part of the colon.

Mr. Lane has apparently also experienced this difficulty, for he has recently excised the colon in whole or in part at a subsequent operation.

In cases of my own I have found that a large high soap and water enema introduced slowly once a month obviated this complication, and should this fail in any case I should perform the simple little operation of appendicostomy and irrigate the colon from above at regular intervals rather than undertake so extensive an operation as excision of the colon.

March 9th, 1908.

Dr. J. E. Moore considers that thirst is the greatest source of suffering after operation, and in the management of this symptom many mistakes are made. His patients all get water after operation, and they convalesce much more comfortably than those who are deprived of it. All surgeons agree that the patient is better without food for a time after operation, for he does not suffer from hunger, and an effort to force feeding disturbs his stomach. Aside from opiates and mild cathartics, medicine has very little place in post-operative treatment. His patients get it only to meet definite indications. Attention should be given to the relief of pain after operation. The proper care of the bowels after an operation has much to do with the patient's comfort. In every case the question naturally arises—When shall the patient get out of bed? All were agreed that old people should get up at the very earliest period possible for reasons well understood, but there was still a great diversity of opinion as to when a younger patient should get up. Every abdominal operation was of sufficient importance to justify the patient in remaining in bed for from one to three weeks.—*Med. Rec.*, vol. lxxiii, No. 7.

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A CLINICAL LECTURE

ON

NEW GROWTH OF MEDIASTINUM AND BRAIN PRESENTING SOME UNUSUAL FEATURES.*

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GENTLEMEN,—The subject of my lecture to-day will be a case, which you saw recently in No. 1 bed of the Arthur Jackson Ward, in which the question of diagnosis and, consequently, of treatment was, for a time at least, of some uncertainty, and in which there was a curious incompleteness in the symptomatology which deserves your consideration. As you know, the patient died, and an autopsy confirmed the diagnosis in all particulars. To-day, therefore, we are able by means of the diseased organs now before you to see the details of what, during his lifetime, could only be conjectured more or less generally.

The case presented itself in two distinct parts, which, though connected with and dependent upon each other, were yet accompanied by such distinct sets of symptoms that we may consider them at first separately.

The history given by the patient—a plumber, æt. 45 years—is, briefly, as follows: About August, whilst he was at work, he was seized with attacks of twitching in the left arm and leg, of which he was quite conscious. They lasted for a few minutes, preventing him from working, then passed off. He thought they corresponded with his heart beats, which were violent and sensible during the attacks.

He continued at his work for about a month, but after that had to lie up on account of the recurrence of these attacks and increasing weakness in the left leg.

In October the left arm and leg became completely paralysed, and have remained so ever since. For this left hemiplegia he was admitted to hospital in November. These were all the symptoms of which he complained on admission. He was perfectly intelligent and clear, and denied having had any headache, vomiting, or loss of sight.

On examination there was a typical left hemiplegia with some rigidity, marked increase of myotatic irritability, and no evident sensory changes.

An examination of the eyes showed marked swelling of the discs on both sides. This fact, together with the history of the onset of symptoms, made the diagnosis of an intra-cranial tumour affecting the right Rolandic area highly probable. At first it caused symptoms of irritation, namely, attacks of focal or Jacksonian epilepsy—localised convulsions without loss of consciousness. As the tumour increased in size the nerve-cells, which had previously been irritated and stimulated to over-action, became squeezed more and more, causing at first paresis, later paralysis. Secondary degenerative changes followed in the damaged nerve-tracts, causing muscular rigidity in the parts supplied, together with increased myotatic irritability.

So far all is simple and easy to follow. You are, however, taught to expect three so-called cardinal symptoms in cases of intra-cranial tumour viz. headache, vomiting, and optic neuritis. In this case, as you see, two out of those three symptoms were absent throughout. That being so, was the diagnosis of intra-cranial tumour warranted? The answer is, it certainly was warranted in this case, because of the additional presence of a fourth cardinal symptom, namely, the hemiplegic convulsions. Had there been no Jacksonian epilepsy, then the absence of two out of the three cardinal symptoms mentioned above would have made a diagnosis of intra-cranial tumour somewhat bold. When, however, double optic neuritis and localised convulsions, followed by hemiplegia, are both present the diagnosis is practically certain.

As a matter of fact headache in cases of intra-cranial tumour is variable in degree. Sometimes it is extreme, agonising; in others it is severe at times, in others moderate only, whilst only rarely, as in this man, is it absent throughout. This variability does not depend upon the size of the tumour. In this case (see Fig. 1) it was of large size yet painless, whilst I have seen most severe headache occur where the tumour has not been larger than a marble. Nor does it appear to

* Delivered at the University of Sheffield.

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depend on the rate of growth. In this man the whole illness was only of six months' duration, during which the growth must have been fairly rapid, and yet there was no headache.

We really know little as to the actual causes of headache in cerebral tumour. But it is worth while to remember that you have seen a case in which a fairly large rapidly-growing tumour occupying the superficial and deeper parts of the right hemisphere in the fronto-parietal region has run its course *without causing headache.* The liability to headache in various individuals is variable under all circumstances. Some persons never know what it is to have a headache, whatever is the matter with them; others do on the slightest provocation. For example, in the early stages of typhoid fever headache may be intense, or it may be absent throughout, and so also in early syphilis, and in plumbism.

As regards vomiting, its absence may be associated with the absence of headache. In many cases the vomiting only occurs at the height of the paroxysm of headache, and the severity of the latter seems to cause the former. In this respect we have a similar series of events in the ordinary sick headache or migraine.

Apart from that, however, it is generally accepted that vomiting is less marked in tumours of the hemispheres than in certain other parts, notably the cerebellum.

On removing the brain it was noticed that the right hemisphere over and behind the upper part of the Rolandic area was distended, the convolutions were flattened, and the sulci more open and shallow. This part also felt softer than normal to the touch, and gave a feeling of fluctuation. At its upper part along the upper edge of the longitudinal fissure a hard nodular area about one inch across was seen; it was slightly depressed below the surface, the pia mater adherent and thickened, convolutions absent.

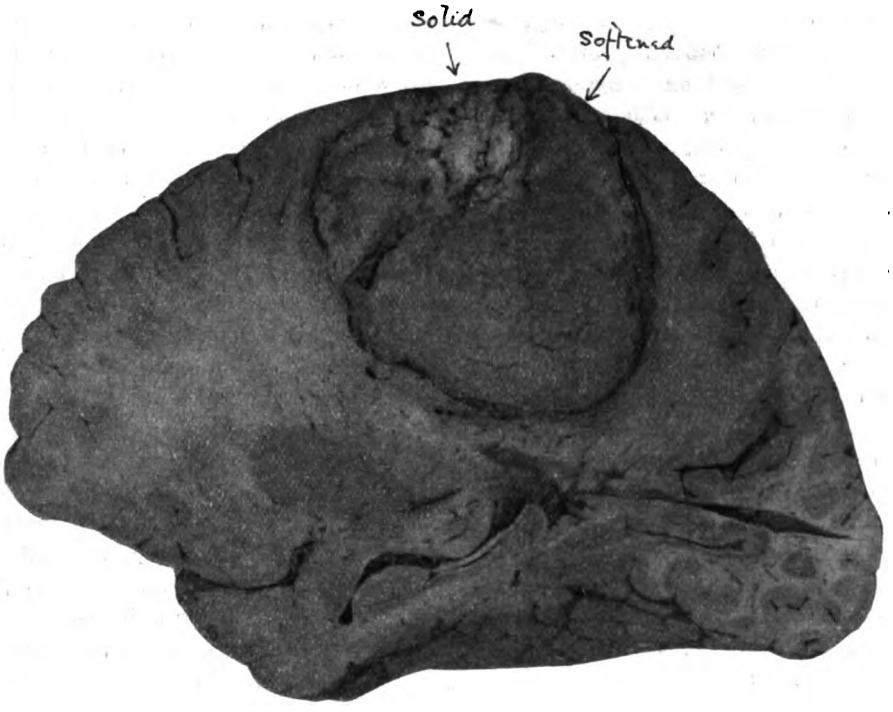

FIG. 1.—Large encapsuled endothelioma. Right cerebrum. Vertical longitudinal section.

After hardening the specimen a vertical longitudinal section was made about half an inch to the right of the longitudinal fissure (Fig. 1) This passed through the centre of the tumour, which extends from the upper surface of the brain downwards for about two and a half inches. It is completely shut off from the brain substance by a

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capsule. The only solid portion is at the upper part, the rest being a semi-gelatinous, yellow, softened mass.

The tumour has evidently caused symptoms rather by pressure than by infiltration. Structurally it is an endothelioma.

We have as yet dealt with but one part of the case; let us now take up the subject once again from the date of his admission to the hospital, in the month of November. You will remember that he complained of no symptoms except those connected with the cerebral tumour. On looking at him, however, his appearance strongly suggested the presence of an intra-thoracic growth. The face was slightly cyanosed and congested, the veins fuller than normal, the ocular conjunctivæ congested. This facies, when present, is almost pathognomonic of intra-thoracic growth; in its typical form, I think, it differs from that of aneurysm, or of any form of heart disease. There is a suggestion that the patient is being slowly strangled without the intense lividity which accompanies a similar state in right heart failure.

The arms were found also to be swollen, the left more than the right. There was marked dilatation of the cutaneous thoracic veins, whilst coursing down the abdomen on both sides there were prominent enlarged superficial veins in which the blood was running from above downwards.

Obviously, therefore, the man had some obstruction of his superior vena cava. The alternative superficial abdominal route for the venous blood is thus made use of whether the superior or inferior vena cava is blocked. If the latter, the current in the superficial epigastric vein is reversed by the increased pressure in the femorals, and the blood flows from below upwards—that is, in the same direction as the stream in the blocked cava; whilst if the superior vena cava is obstructed, it is the stream of the internal mammary veins which become dammed back, and the direction of blood flow in the superficial veins follows the direction of that in the blocked superior cava. On examining the chest the diagnosis of an intra-thoracic new growth was fully confirmed by the abnormal physical signs in the upper front of the right lung. Here was found an area of dulness below the right clavicle extending to the edge of the sternum, outwards to the middle of the clavicle, and downwards to about the third space. There was diminished expansion, the breath and voice sounds were also diminished over this area. The rest of the right lung appeared perfectly normal, so far as could be detected by physical signs. The heart was not displaced. The most curious feature of the case during life was the complete absence of subjective symptoms from this intra-thoracic growth. He absolutely denied any pain, cough, or dyspnœa up to, or on, admission. Nor did he complain of any pain during the weeks in hospital prior to his becoming comatose. Similarly he had *no cough or sputum at any time.* As to the absence of subjective dyspnœa, even if it was too slight to trouble him, yet there was notable frequency of respiration even from the first, whilst lying in bed. This varied from 25 up to 32 or more per minute.

Later on, when he began to be drowsy and stuporous, there were several attacks of dyspnœa which almost proved fatal.

If we now turn to the post-mortem findings, you see that there is a very large mass of growth occupying the upper lobe of the right lung. In parts it is hard and dense, whilst the greater part of its centre is completely softened and broken down. It has not infiltrated the lung substance by spreading alongside the branches of the bronchial tree, as is commonly seen in mediastinal new growths, but rather to have simply pushed the lung tissue in front of it, so that there is a sharply-defined margin between growth and lung, the latter being compressed into a thin layer, especially near the apex. This growth measures about five inches in diameter, is rounded, and seems to be a very much enlarged malignant peribronchial gland. Several other smaller, but still enlarged, malignant glands are situated about the right bronchus and lower trachea.

Microscopically the tumour is an endothelioma.

No other organs in any part were affected with the disease.

You will remember that during the patient's life I frequently pointed out, not only the absence of all cough and sputum, but also the fact that there was no evidence of any pleural effusion at the right base. Such pleural effusion is quite common in cases of mediastinal new growths where the growth involves, as it usually does, the vessels at the root of the right lung. Often it is one of the earlier signs of the new growth's presence; at other times it does not appear till later, but it

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comes sooner or later in most cases. Another feature to which I called your attention was the apparent absence of any progress in the thoracic growth, the physical signs remaining absolutely the same from the time of his admission up to death—a period of about three months. These negative features, the absence of cough and sputum, of pleural effusion, or of increasing physical signs, led me to suggest that probably the tumour was small, and that it had not involved the root of the lung in the usual manner. Of these two surmises, a glance at the specimen will show you how wrong the first one was.

The growth is distinctly large; the second suggesting that it had not affected the root of the right lung to any extent proved more correct. The growth has not spread in that direction, but rather in the direction of the upper lobe.

Whilst, therefore, the mass of growth is considerable, it has remained more or less encapsuled. If you compare it with any of the other specimens in the Museum you will notice this difference.

As regards the pressure on the superior vena cava, the specimen shows how much it was squeezed by the growth. This symptom is one of the most characteristic distinctions in the differential diagnosis of intra-thoracic aneurysm and new growth. This is easily explained: An aneurysm increases merely by expansion; when it comes in contact with such a structure as the superior vena cava, the latter becomes slowly and gradually stretched over it, remaining for the most part patent and capable of carrying on the circulation. But a new growth coming in contact with a vessel grows round it on all sides and speedily chokes or squeezes it so as to cause more or less complete obstruction. On the whole, then, we may say that early and marked signs of superior vena cava obstruction favour the diagnosis of new growth rather than aneurysm.

But in whatever manner an intra-thoracic tumour begins, it is perfectly certain that it will not be recognised in its very early stages. No one can make a diagnosis without sufficient data to go upon; he may make a guess, a successful shot, but that is all.

Sometimes it happens that in the early stages there is slight hæmoptysis with cough, and the case is diagnosed as phthisis; at other times the accompanying pleural effusion is diagnosed as simple and primary; in fact, the errors that must occur before definite signs show themselves are numerous.

As to the diagnosis of this particular case, I have said enough to show you that the physical signs were unequivocal, whilst the absence of several subjective symptoms both in the thorax and brain was curious and unusual.

As to treatment, we need say nothing of the thoracic condition; it was beyond any form of treatment known to us as yet. The brain lesion, however, was a different matter, and, if standing alone, would have been eminently a case for surgical operation. Moreover, as the autopsy showed, such procedure would most probably have been successful. Seeing, however, the probability that both lesions were malignant, also that the intra-thoracic obstruction might prove rapidly fatal on the administration of an anæsthetic, we decided that any severe or prolonged cranial operation was more likely to hasten death than to afford any reasonable hope of improvement.

March 9th, 1908.

GOGGIA calls attention to the fibrillary contraction which occurs in the brachial biceps when the belly of the muscle is struck suddenly but lightly and is then pinched gently between thumb and finger. The aim is to apply a sudden, brief, and comparatively gentle mechanical stimulus to the muscle. It induces a sudden local contraction of the belly of the muscle at this point, when the individual is debilitated from any cause. Under normal conditions the contraction extends throughout the whole muscle and does not remain limited to the point irritated. This sign has been described in the literature under the name myœdema or idio-muscular reflex, but it has been regarded as a sign of any debilitating disease. Goggia affirms, however, that while it is constant in typhoid it is present only exceptionally in other diseases. It was positive in his 18 typhoid patients, also in 11 out of 18 pneumonia patients, in 2 out of 9 with erysipelas, and in 3 out of 4 with grip, but the reaction was much more pronounced in the typhoid patients than in most of the others, and he thinks that it can be accepted as a valuable adjuvant sign of typhoid fever.—*A. M. A. Journ.*, vol. l, No. 8.

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WITH MR. CORNER IN THE OUT-PATIENT DEPARTMENT OF ST. THOMAS'S HOSPITAL.

A Case of Fracture of the Coronoid Process of the Ulna.

This patient, a man, comes with a condition in his elbow which he said appeared on November 11th, and he tells us that he fell on his elbow five weeks previously, at which time he did not think he had hurt himself. At the end of five weeks he came here, and when I saw him there was very little in the way of physical signs. So he illustrates a kind of case that has become of far greater importance in these modern days than it was in the time of our predecessors. It is an obscure fracture which resulted from an apparently trivial injury. These obscure fractures our forefathers never used to diagnose, but since we have the use of the X rays we can recognise many of these conditions. Before the era of X rays we should not have been able to get the least idea of what this was. We had this man skiagraphed, and found that the injury which he received was a fracture of the coronoid process of the ulna. That, as you know, is a very uncommon form of fracture, except when it is combined with backward dislocation of both radius and ulna at the elbow. This man certainly never had backward dislocation of both bones at the joint, or he would have had to consult some medical man before he came here. So here is an opportunity of examining what is a very uncommon condition of affairs. It is noteworthy that in this fracture there is very considerable limitation of movement, the arm going through only about 90°. And not only is there limitation of flexion and extension, but you also find there is some limitation of pronation and supination. So this was not a case of fracture of the coronoid process of the ulna alone, but there is also an injury to the elbow-joint. If there were not an injury to his elbow-joint, there should not be this limitation of movement. It is now four months since the accident, and he has been having massage and movements ever since. In these cases you are liable to find some limitation of movement, because the coronoid process is broken off and held away from the ulna by the brachialis anticus muscle, which is inserted on it. So that you never find bony union after this fracture at all; you only get fibrous union. And at the same time, in fracture of the coronoid process, you will always get a tearing of the anterior ligament of the elbow-joint, and always a certain amount of adhesions and blood clot in the elbow-joint. It is their results which cause the limitations of movement. As massage and manipulations have been found to be insufficient to correct these restrictions of movement, there is only one further thing left, and that is, to give him an anæsthetic and see if the movements which are desired can be performed—that is to say, flexion, extension, and rotation at the elbow-joint.

A Case of Flat or Abducted Feet.

The next patient is another man who has come from the Physical Exercises Department of the hospital, and you can see that he has flat foot. He is instructive to you in the way that he illustrates the fact that the text-book treatment of exercises for flat foot—that is to say, strengthening the muscles of his calf—has not cured him. He has been attending the Physical Exercises Department for seven weeks, and yet he has not improved. One comes across these cases of flat foot which do not improve with physical exercises, and they teach two things: First, that people who have flat foot are more prone to suffer from injuries to their feet than are people who have arched insteps.* If a person has a normal foot with arched instep he seldom has any injury to his foot; whilst the persons with flat foot, if they fall or make a false step, are very likely to injure their feet. The second thing that they teach you is that a flat foot is not simply a case of decrease in the arch of the foot. It is really the result of a distinct alteration in position. In an ordinary arched foot the toes are what is called adducted, the movement taking place at the tarsal joints. A foot may be temporarily flat and abducted, in which case the arch can be restored. But if his foot is permanently flat it is permanently abducted. When the bones have become inured and fixed in the position of abduction, the condition of flat foot becomes permanent, and no amount of tip-toe exercises will cure it. In dealing

* "The Position, Deformities, and Injuries associated with Adduction and Abduction of the Feet," Clinical Journal, November 27th and December 4th, pp. 105–112, and 120–128.

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with these two great positions of the foot—abduction and adduction—for the sake of memory you need only remember two rules. It is the tibialis posticus which is the muscle that adducts the foot and raises the instep. It is the peronei muscles which abduct the foot and depress the instep. You will easily see why it is that people whose feet are permanently flat do not improve with physical exercises. The reason is, when the foot gets fixed in the abducted position the muscles which formerly abducted the foot cannot abduct it further. It is as if a pump was used to lift a weight, but if the weight is already lifted the pump can do no more. It is similar with the peronei muscles; they used to abduct the foot, but when the foot is already abducted they cannot abduct it any more. When the foot is permanently abducted, the peronei become merely flexors, like the other calf muscles. Such a man is often taught to carry out tip-toe exercises, and those exercises strengthen the muscles of his calf, of which the peronei are now one set. So that these exercises which are supposed to cure the flat foot strengthen the very muscles which prevent the foot becoming otherwise than flat. So you will find any number of these cases in which you follow the text-book treatment of teaching tip-toe exercises, and yet they do not improve at all. When that is the case there is only one thing left. You have to remember that the feet are permanently flat, and that condition you cannot alter. And as you cannot alter it you have to make the best of it, exactly like Mahomet and the mountain. And the only way to make the best of these things is to give the man a pad under the arch of his foot. Then he will be relieved. It is the same complaint with these people, whether they have these abducted or flat feet, or whether they have had a fracture of both bones of the leg, that the thing which they cannot do is to go up a ladder or along planks. And some of the most distressing cases which you will find are those of flat foot which result from a badly-set Pott's fracture. There is hardly any other fracture in the body which will detract so much from a working man's working value as a Pott's fracture, or any other fracture which leads to flat or abducted feet.

A Case of Hydrocele after a Syphilitic Testicle.

This man comes to us with a swollen testicle. He is æt. 28 years. Five years ago he had a sore on his penis, which was followed by a rash and sore throat, so that there is no doubt about his previous syphilitic infection. Nine months ago he came here with some swelling of his testicle, which went down and disappeared after he took medicine (iodide of potassium), and now he has come up again with swelling of the same testicle. So that, by relying on the history, there would be no doubt that the diagnosis of a gummatous disease of the testicle would be made. But a superficial examination shows that there are differences between the patient's condition and that of gummatous disease of the testicle. The first of these is that the swelling on this occasion is tender. As you know, a gummatous testicle should have no tenderness. The swelling is smooth in outline, and not hard and heavy as a syphilitic testicle is said to be. Further, if one tries the test of translucency one finds that four fifths of the swelling in the right side of the scrotum, in this man's case, is hydrocele, and that the testicle is little enlarged. Still, if that hydrocele is dependent on syphilitic disease of the testicle it ought to clear up under anti-syphilitic treatment. And herein comes the clinical importance of this particular case. Nine months ago it is quite clear that he had a tertiary syphilitic disease of his testicle. But it is more than likely that now he has not tertiary syphilitic disease of his testicle, but he has a chronic inflammatory condition of the tunica vaginalis left behind as the result of the syphilitic disease. If that is the case the cause of the hydrocele is not syphilitic, and in all probability it will not clear up under anti-syphilitic treatment. We will start him on anti-syphilitic treatment, and if it does not begin to clear up quickly we shall have to treat him as a case of hydrocele.

A Case of Fractured Clavicle.

This boy, æt. 12 years, comes to us, having fallen off a swing just over a month ago. His clavicle is broken by the indirect violence of the fall on the shoulder. At this point, you will remember, is the weakest point of the bone, at the junction of the two curves. What about the boy's condition? One knows that fractures of the clavicle at the age of twelve will get well. Why? This has united all right. As the boy grows up, so the growth of the clavicle will tend to restore the bone to its original shape. There is a very great difference in the

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prognosis of fractures of the clavicle in the growing subject and fractures of the clavicle in the fully-grown subject. Probably when he is twenty-one there will be no sign of it left.

A Case of Repeated Unilateral Dislocation of the Lower Jaw.

This young man, æt. 21 years, comes to us because his jaw has several times slipped out of the joint on the right side, when he cannot properly shut his mouth. If he yawns and gapes it dislocates the right side of his jaw. Why? Because when he opens his mouth two convex surfaces, the condyle and the eminentia articularis, are up against each other. And the dislocation is unilateral because very few people open their mouths perfectly straight: watch people singing in church, they exert the muscles on one side of their face to a greater extent than those on the other side. And so whenever you come across these cases of habitual dislocation of the jaw, the accident occurs on one side and not on both. What structure in the temporo-maxillary joint tends to keep up this condition? It is the intra-articular cartilage, and that cartilage has two parts—a thick and a thin part—over the eminentia articularis. When a person opens his mouth the condyle of the jaw comes forward. As a result of frequent dislocation the intra-articular cartilage becomes thickened lower down where it originally was thin, so that when the jaw comes forwards it slips more easily off the eminentia articularis and dislocates. One might point, as a parallel, to the dislocation of the semilunar cartilage of the knee. If a person dislocates the internal semilunar cartilage several times it gets into the habit of always slipping out. The jaws are the same in that respect. If a person dislocates one side of his jaw two or three times, then he gets into the habit of always doing it. The treatment for dislocated semilunar cartilage is to operate and remove the cartilage, but we have not yet quite arrived at the stage when we remove the cartilage from the temporo-maxillary joint. He tells us that two years ago his jaw came out, and it has done so three times since, and that it has not improved in the meantime. There are two ways of treating these patients—one is by means of rest, and the other is by operation. One will naturally first try the rest treatment. He must rub his face well over the condyle and avoid opening the mouth wide. He says the first time the dislocation occurred was while he was in bed at night. He was fully awake. The most difficult cases to treat are those in which the person yawns when he is waking up, before he has collected his scattered senses, and the dislocation has happened before he quite realises it. Those patients should wear a four-tailed bandage, as for fracture of the jaw, at night. One can persevere with this treatment for any time from a year to two years. In the majority of cases the condition settles down and simply ceases to occur. There is one thing you always want to guard against in this condition, and that is the possibility that there may be a fractured base. If there has been a fractured base and it has gone through the foramen ovale, you can get paralysis of the muscles supplied by the nerve that goes through that foramen. These will be chiefly the pterygoids. If the muscles on one side get paralysed the corresponding muscles on the other side may dislocate the jaw. As a rule it is easy to exclude fracture of the base, because if a man has had a blow on the head sufficient to badly fracture his base it is obvious there will be other signs and there will be a history of it. This man has neither history nor sign of that, so we can exclude it.

An Irreducible Umbilical Hernia.

This man, æt. 42 years, a stout subject, comes with a huge umbilical hernia. You know that these umbilical herniæ are infinitely more common in women than in men. This is quite irreducible. He tells us that he has had the hernia since last March, and that it came after he had been lifting. You can feel that the contents consist of both bowel and omentum. You must always be prepared to find that these herniæ are really bigger than they look. One night I had to go down to see an old lady who had a more or less strangulated umbilical hernia. She was enormously fat, so that her general stoutness covered up the whole of this umbilical hernia except its top, which presented quite a nice little point. The man who was looking after her had explained that it was only a tiny rupture, and he thought the strangulation could be relieved under cocaine. But one found in the midst of the fat a hernia as large as a football, which, of course, no one could expect to be done under cocaine. But, fortunately, she did not want anything done in the way of operation, and I was

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glad to get away. This man is only 42; he is a general labourer, and has many years of active work before him, so that there is only one treatment to be recommended, and that is that he should come into the hospital and be operated on. It is his only chance of being able to do hard bodily work again.

A Case of Vascular Spasm.

This woman is æt. about 40, and comes with a finger presenting a curious condition. She says it has never been injured. When it becomes cold she suffers excruciating agony, she says, but she can move it quite well. She has been feeding on "nerve foods" since last Easter, but whatever her bodily condition may be the finger persists in this unsatisfactory condition. You see there is some discoloration of the nail, which is brittle and has grown out of shape. When that is the case the thing one suspects is that there is some nerve lesion. That lesion may be either local in the finger, or anywhere as high as the shoulder, where the nerves which supply that finger go to form part of a bigger nerve trunk. But you would expect in that case to find many of the other fibres of that trunk involved. In this case there is no sign or history of that. So if it is a nerve lesion it is obvious it must be a local one. Another feature which leads us to think that it is not a nerve lesion is the course which she says the pain pursues. As a rule, the pain due to a nerve lesion follows fairly accurately the course of the nerve, and that nerve is tender to the touch. As you know, the ulnar nerve supplies both the palmar and the dorsal surfaces of that finger, and her pain is not referred in any way along the course of the ulnar nerve. So I assume that the trouble is not a nerve one. If we adopt that idea there is only one thing it could be, and that is something connected with the vascular mechanism. And there one thinks of such a condition as local asphyxia or Raynaud's disease, which produces blue hands and ears, and so on. In other words, I regard it as a vascular spasm. There is also, of course, hyperæsthesia. She tells us that when the pain is very great there is considerable relief from immersing the finger into very hot water. She keeps the finger wrapped up, both summer and winter. Of course, the great difficulty arises in treating these things. She has had the trouble twelve years, and in that time it is likely she has tried every variety of palliative treatment which could be thought of, so the question before us now is as to whether we shall recommend anything in the way of radical treatment, which would consist in taking away the finger. Her pain is not due to ingrowing nail. In the case of ingrowing toe-nail the pain is due to septic ulceration underneath, but there is no question of that here. If the trouble is connected with the nerve I am not sure that removal of the finger will cure it permanently, though probably it would do so for a time. Before that, I think, we will advise that the finger be painted with iodine, and we will give her some nitro-glycerine to take. The iodine acts as a vaso-dilator. If that does not answer I shall advise her to have it removed.

A Case of Sebaceous Cyst.

The last patient we can see together this afternoon is a woman, æt. 34 years, who comes with a superficial swelling near the left ear, which is one of two things: either a sebaceous cyst or a lipoma. What is the great distinction between the two? I remember, when a student, incurring the displeasure of a surgeon because I suggested a "lipoma" looked as if it was pointing. And it was. The difference is that a lipoma is under the skin, and a sebaceous cyst is in the skin. You cannot pick the skin up over this, and therefore it is a sebaceous cyst which has solid, not fluid contents. Of course, a lipoma may become inflamed and adherent to the skin, but that is not usual.

March 9th, 1908.

THE ADMINISTRATION OF THE NAUHEIM TREATMENT.

Much has been said and written of late about the administration and efficacy of the artificial Nauheim Baths in England. That good results similar to those obtained at Bad-Nauheim itself are possible the bulk of evidence goes to prove. Indeed, Dr. Schott, of Bad-Nauheim, has declared this to be the case. The generally accepted theory of the action of the Nauheim Baths may be briefly defined as follows: The stimulation of the nerve-endings by the action of the CO_2 given off causes dilation of the peripheral circulation, so causing a larger arterial "bed," and at the same time reducing the amount of work of the central cardiac mechanism. Here comes the important factor, namely: This peripheral dilation is produced

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without raising the temperature of the baths. The effect of a raised temperature in dilating the capillary area of the body (as in the case of the ordinary hot bath) is well known. But this is accompanied by disturbance of thermogenesis, which is both injurious and exhausting, as it increases tissue waste. But, whatever the theory, experience undoubtedly teaches that the effect of the Nauheim Baths in slowing the pulse-rate, decreasing the size of the heart, and allaying nervous irritation, is an indisputable fact, and, further, that this result is more permanent than the effect produced by digitalis and allied drugs.

Simple cardiectasis, so-called nervous heart, and myocarditis are undoubtedly the affections that respond best to the treatment. But slight organic valvular lesions are apparently cured, and there is no doubt that patients suffering from pronounced valvular disease are often entirely freed from the symptoms.

Rheumatic cases and certain forms of nervous disorders are also treated with excellent results.

When good results have not been obtained the fault is generally traceable to the improper administration of baths and treatment. Unfortunately, the introduction of the mineral ingredients of the baths in the form of soluble tablets has in many cases led the laity to experiment with a method which thus recommends itself as being so easily and cheaply available. Such unskilled experimenting in the cases of cardiac sufferers is undoubtedly open to criticism, and could not fail at times to produce questionable results.

The lack of result in many cases is due to this imperfect method of attempting to produce the characteristic Nauheim effervescence. In the tablet-prepared baths the gas is not formed and distributed homogeneously, but in patches.

The other and rarer method of forcing the carbonic acid gas into the bath under pressure is open to a similar objection, as it is impossible to retain the gas in solution unless it has been for a considerable time under great pressure, in the presence of the vehicle.

A method of preparation is necessary which more closely approaches the natural "champagne" baths at Bad-Nauheim. As a well-known physician said when asked his opinion of a so-called Nauheim bath as prepared at a well known "hydro": "It is as much like a Nauheim bath as lemon kali is like champagne."

However, it is a hopeful sign of the times that at last a genuine attempt has been made to give the Nauheim baths and treatment in a scientific way, at the new St. George's-Nauheim Institute, at 6 and 7, George Street, Hanover Square, London. This Institute has now passed under the control of the medical profession. This is certainly a matter for ethical congratulation, as the establishment is an acquisition to the profession.

Some idea of the capacity of the institution may be gathered from the mention of the fact that it provides accommodation for over one thousand baths per week.

The baths are the nearest approach to the real thing that has yet been done. A continued effervescence, lasting the whole of the duration of the bath (up to twenty minutes,), constant and homogeneous in character, is obtained. Imported Nauheim "mother-lye" is used in their preparation, while the saline properties are obtained from Droitwich. All the various materials used in the formation of the different baths, are carried to the bath-rooms in solution by means of a system of pipes which draw their supply from large tanks on the roof. The bath attendants, in preparing the differently constituted baths, follow strictly the prescriptions of the physician-in-charge. The bathing arrangements are good and the cubicles are excellent, being furnished with couches on which the patient takes his prescribed period of rest.

The management have been at some trouble, in their effort to make the treatment as complete as possible, in arranging for the inclusion of the Nauheim drinking waters as part of the treatment.

In the present policy of the institute great care is taken to insure that the recommending doctor retains his patient. The physicians-in-charge of the Institute particularly request his attendance for consultation in regard to any suggestion as to any particular line of treatment, reserving to themselves only the actual writing of the prescription. The recommending physician's name and address appear on the prescription form and on the chart of treatment. This chart of treatment should be of particular use in the collection of statistics, as it contains full details of pulse, arterial tension, exercise, massage, rest, etc., before and after each bath, while on the notes appear the area of cardiac dulness, and anything of consequence that the physician-in-charge may from time to time observe. This will shortly further be amplified by the installation of the orthodiagraph. A copy of the chart and a weekly report are sent to all physicians who have patients under treatment.

It is pleasing to note that this enterprise is entirely English in character.

There is to be an element of useful philanthropy in the Institute, for at the request of the medical officers, the directors are about to make an offer which will place the benefits of the St. George's-Nauheim baths and treatment at the disposal of the poor. This offer is to be made to the general hospitals of London, each of which will be invited to have one patient under treatment continuously. As one patient finishes his course another will be at liberty to commence. In this way there will be some eighteen or twenty patients under treatment at the same time.

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THE CLINICAL JOURNAL,

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE, AND CLINICAL REVIEW.

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A CLINICAL LECTURE

ON

A CASE OF ADDISON'S DISEASE.

Delivered at Guy's Hospital.

By W. HALE WHITE, M.D., F.R.C.P.,
Physician to Guy's Hospital.

GENTLEMEN,—Here is a very remarkable thin quarto book, and it is one which I think ought to make us all feel proud, because much of the fame of this hospital rests upon it. It is by Addison and on disease of the supra-renal capsules; in this small compass two entirely new diseases are described. And they are described not as the result of any experimental observation, but solely because Addison, by his power of observation, was able to make out that these two new diseases existed. I think if you will reflect on how difficult it is to observe properly, you will agree with me that the publication of a book like this is one of the highest flights of the power of observation carried to the point of being real genius. As we have in the wards at the present time an instance of both of these diseases which are described in this book, I thought we could not do better than talk about Addison's disease this afternoon. The date of the publication of this book is, as you will see afterwards, 1855. Many years before Addison published this book, the full title of which is, 'On the Constitution and Local Effects of the Disease of the Supra-renal Capsules,' he read a paper at the South London Medical Society on the same subject, and waited some years to see if his views were correct, and then he finally published this famous book. Perhaps I might read to you his account of the disease, and then we will see how far our patient fits in with it. In my own copy—because unfortunately, the first edition, of which this is a copy, is rare, and I have not a copy myself of that

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edition—I will read you what he says: "The leading characteristic features of the morbid state to which I would direct attention are anæmia, general languor and debility, the remarkable feebleness of the heart's action, irritability of the stomach, and a peculiar change of colour in the skin, occurring in connection with the diseased condition of the supra-renal capsules. The patient, in most of the cases I have seen, has been observed to gradually fall off in general health, he becomes languid and weak, indisposed to either bodily or mental exertion, the appetite is impaired or entirely lost, and the whites of the eyes become pearly, the pulse small and feeble, but excessively soft and compressible. The body wastes, not, however, presenting the dry shrivelled skin and extreme emaciation usually attending malignant disease. Slight pain or uneasiness is from time to time referred to the region of the stomach, and there is occasionally actual vomiting. Notwithstanding these unequivocal signs of feeble circulation, anæmia and general prostration, neither the most diligent inquiry nor the most careful physical examination tend to throw the slightest gleam of light upon the precise nature of the patient's malady, nor did we succeed in fixing upon any special lesion as the cause of this gradual and extreme constitutional change." Then he goes on to describe the pigmentation, but that I will do when I tell you about our own patient. You see he gives a distinct clinical picture of the disease. And then the greatness of his discovery lay in the fact that he pointed out that invariably, when those symptoms were present, disease of the supra-renal capsules was found. You notice that there are three or four very important signs which can be picked out of his description, and although the pigmentation is not the first of the symptoms to appear, it is so much the most important that I thought we would take that first. I am sorry to say that I cannot bring you the patient here to-day because in the last few days he has become much worse; it would probably kill him to bring him here. We could hardly feel his pulse this afternoon. If you will go and look at him in the ward after the lecture you will see that all over he is stained dark brown, and that here and there there are darker patches on the top of that dark brown. Addison described it as "a dingy or smoky appearance, of various tints or shades, of deep amber or chestnut brown. And in one instance the skin was so universally and deeply darkened that, but for the particular features, the patient might have been mistaken for a mulatto." And that really describes our patient as well as any words of mine. As I cannot show you the patient I have brought you Addison's original coloured pictures. The first shows a case with rather light colouring, but still it is the tint you expect to find in Addison's disease, and it is the tint which you will notice in our patient. Here is another picture of an even fainter tint. Those pictures give you a very good idea of the tint observed. There are a few points worthy of note about pigmentation. There are certain places in the body where it is darker than elsewhere. It is darker where the skin is inclined to go into folds naturally, so it is especially darker in the axilla, as shown in one of these plates. It is especially dark over the genitals. In the scrotum and in the penis against the thigh there are a good many folds of skin and points of contact, so that pigmentation is always very dark there. And if you pull down the foreskin and look at the glans penis you will often find pigmentation there too. Our patient shows it admirably. It is often very striking on the nipples; notice how dark the nipples are on that plate which Addison published. And it may be seen especially in the flexure of the knees, and it is very dark wherever there is a band round the body, so that if a man has worn a belt he has a rather darker band round his body. Also if a man's chest is examined, where his braces have pressed, the pigmentation will be darker there, and the same is observed where the garters have been on the legs. And lastly, it is usually darker where there are any prominences, such as acromial prominences, for instance. You may see it anywhere inside the mouth. Our patient has a beautiful patch of it just inside the mucous membrane of the mouth, where the upper and lower lips meet. You may see it on the palate in some patients, and even on the tongue. It has been described as being seen under the nails, but I have never seen a case where the pigmentation was present there myself. Pigmentation has also been observed under the conjunctiva, but I have not seen it there either. People become pigmented from many different causes, and probably all of you have seen cases in which the diagnosis

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of Addison's disease has been discussed, and it has been difficult, perhaps, to come to an opinion at first sight, and so I thought it would be very practical if I mentioned to you the fallacies. I think perhaps the most common fallacy is to mistake Addison's disease for pernicious anæmia, which is the other disease described by Addison in this work which I have shown you. Pernicious anæmia, you know, is a disease in which there is excess of blood destruction, and we have evidence of its destruction in the deposit of free iron in the liver, and also in the large amount of urobilin which is excreted in the urine. In that disease the skin goes a peculiar lemon tint, probably being stained by some of the products of the abnormal blood destruction. This yellow staining is quite different from the characteristic case of Addison's disease, but in slight cases confusion often arises. Nowadays confusion could hardly arise if you had the patient under protracted observation, because examination of the blood in Addison's disease shows very little abnormal, and examination of the blood in pernicious anæmia shows you that there is a very considerable degree of poikilocytosis, and there are megaloblasts, there are nucleated red cells, and the colour index is raised. But let me here again remind you, as I am so often reminding you at the bedside, that you must not be content with negativing the diagnosis of pernicious anæmia because a single blood examination does not show the characteristic changes. The blood in pernicious anæmia varies much from day to day and hour to hour. And often you cannot make a diagnosis of pernicious anæmia on a single examination of the blood only. But if you are able to make several examinations there is no difficulty in distinguishing between pernicious anæmia and Addison's disease, quite apart from the colour, which, indeed, is usually quite different in the two diseases, lemon-yellow in pernicious anæmia, dingy-brown in Addison's disease. Another disease about which there is often great difficulty is to tell whether a patient has Addison's disease or whether he has had his skin stained as the result of prolonged administration of arsenic. You will, I am sure, agree with me that that difficulty frequently turns up in the wards, and some of the senior men here may remember that a year or two ago there was in the hospital under my care a man who was very considerably pigmented, and there was a constant discussion as to whether or not his pigmentation was due to Addison's disease, whether or not it was due to arsenic, or whether it was due to pernicious anæmia. You will remember that the point of interest about that man was that he had some pigment patches inside his mouth. At the time I did not know whether or not the pigmentation of arsenic could ever occur inside the mouth, and we left the matter in doubt; but we came to the conclusion from the examination of the blood—and it is of interest that in only some of many examinations made by Mr. R. W. Allen were the characteristic changes of pernicious anæmia seen — that he had not Addison's disease, but that he had pernicious anæmia, and we were unable to say whether the pigmentation in the mouth was due to his pernicious anæmia or to his arsenic. I took the case before the Association of Physicians of Great Britain and Ireland, which met in London last spring, and fortunately some members present had worked at the outbreak of arsenical poisoning in Manchester, which followed drinking beer impregnated with arsenic. And then I learnt that those who had had such a large experience of arsenical poisoning had never seen a case of pigmentation of the mucous membrane of the mouth through arsenic, and I was therefore driven to infer that the pigmentation in that case was due to pernicious anæmia. But otherwise the pigmentation of arsenical poisoning is very like that of Addison's disease. But remember that the occurrence of pigment in the mouth will be a great point in favour of the case being Addison's disease. Here I show you a picture of arsenical pigmentation, and you will see how similar the tint is to that of Addison's disease. Another cause of difficulty sometimes met with is at first sight to tell the pigmentation which people get who have malignant disease from that which occurs as a result of Addison's disease. But that really ought not to bother you long, because a patient with Addison's disease is not so excessively wasted as is the patient with malignant disease, and in the latter case you will usually find evidence of malignant disease somewhere in the body. Again, in the filthily dirty patients whom you see in hospitals and who are infested with lice there is a pigmentation of the skin from the chronic irritation, but that ought not to give rise to

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much difficulty. Occasionally you see phthisical people with pigmentation of the skin, but there the signs of phthisis are obvious, and that will not cause difficulty. Sometimes after a patient has had syphilis there is a certain staining of the skin, a brown discoloration from the old syphilitic eruption, but I have not seen that give rise to any serious difficulty in diagnosis. And then there is that excessively rare disease, Hanot's cirrhosis of the liver, which is easily detected, with slight jaundice and a big liver, and although the skin is pigmented in that it can give rise to no difficulty. And there is that rare disease called "bronzed diabetes," of which I have never seen an example, in which there is diabetes. But the presence of sugar in the urine will get you out of your difficulty in the matter. The text-books also include the pigmentation of pregnancy, but I have not heard of that being mistaken for Addison's disease. So the differential diagnosis really comes down to this: Has the patient Addison's disease, pernicious anæmia, or arsenical pigmentation? In the last case you have a history of his having taken arsenic, there is the examination of the blood for pernicious anæmia, and so you ought never to go far wrong in the matter. But the difficulty is greater than it seems, because you will meet with patients in the hospital who have been taking arsenic in large quantities at the out-patients' for pernicious anæmia. So much for pigmentation.

The next sign we will speak of, which is the first sign to be detected in Addison's disease, or rather what the patient first complains of in Addison's disease, is an uncontrollable sense of fatigue. Our patient in the ward now has it. We have been looking at him this afternoon. He bitterly resented being disturbed in any way; it wearied him even to turn him over in bed. He is very weak and prostrate, and you will remember that Addison said that the pulse is small and feeble, excessively soft and compressible. The only way in which we have added to Addison's description as far as the weakness goes is that we have of late been taking the blood-pressure with a manometer, and his statement that the pulse is small, feeble and excessively soft and compressible has been amply borne out by the manometric readings. Dr. Mann has been kind enough to take several tracings from our patient in the hospital, and the blood-pressure on the whole has been consistently sinking ever since the man has been in. Here are the readings which Dr. Mann kindly supplied. There are many readings below 60. When the patient came in his maximum systolic pressure was 70, his mean systolic pressure 65, and then shortly afterwards the maximum was 65 and the mean 60. It went up a little then and he seemed to improve, but lately it has gone back very much indeed, and yesterday the lowest pressure had been reached which has been recorded, for the maximum systolic pressure had fallen to 48. That is the lowest pressure Dr. Mann has ever taken, although he has taken so many. And so we have confirmation in figures of Dr. Addison's statement about the smallness and weakness of the pulse. The reason why that should be so I will tell you directly. If you will go into the ward and look at the man you will see that what Addison says is true, that the body wastes without presenting the dry shrivelled skin and extreme emaciation seen in malignant disease. These patients do get thinner, but the wasting is not a prominent feature, and as far as I have seen these patients never get to look like a patient with severe malignant disease. Although they are losing flesh they lose it more slowly than cases of malignant disease do, and the skin never becomes dry and shrivelled.

The last point I mentioned to you that Addison described is that there is slight pain or uneasiness referred from time to time to the region of the stomach, and there is occasionally actual vomiting. That is so in our man. He complains of considerable pain and vomiting. We had him up the other day, but he said he would go back to bed because he felt pain and was sick. There are not many diseases which have fewer symptoms to describe than Addison's disease, and I have already told you all. It is the fewness of the symptoms which prevents this subject being set in examination papers, but that does not make it any the less interesting. Further collections of cases since Addison's death have given us an idea of numerical proportion. It is commoner in men than it is in women, for 65 per cent. of the cases are males. Everyone is agreed that it is very rare in the young and in the old. These patients are usually between 30 and 60 years old.

The greatness of Addison's discovery did not lie so much in the discovery of the disease as that he predicted what would be found if a post-

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mortem was made. He predicted that disease of the supra-renal capsules would be found. Here is a plate from his work, showing the diseased capsules. You will see that the supra-renal capsules have each become converted into an enormous caseous mass. I have brought down many specimens from the Museum; we are very rich in such specimens at Guy's. Here are two huge supra-renal capsules on the top of the kidneys, from a patient with Addison's disease, and they have each been converted into great caseous masses, and I will now pass round many specimens showing the same thing. Of Addison's original cases, which were eleven in number, five showed caseous tubercle in both supra-renals, one showed caseous tubercle in one supra-renal, one showed cirrhosis with atrophy of the supra-renals—and I have brought and now pass round a specimen to illustrate that condition,—and three were examples of secondary malignant disease of the supra-renal, and one, his eleventh case, was a very interesting one: a thrombus had affected the vein going to the supra-renal body so as to lead to impairment of function of the supra-renal body. In this plate in his work you can see the thrombus projecting from the supra-renal vein. Now, to tell you a little more in detail about the changes to be fonnd after death. All collectors of cases are agreed that by far the most frequent thing found post mortem in patients with Addison's disease is that the supra-renal bodies are the seat of chronic tubercle which has gone on to caseation. And we have no doubt that our patient is tubercular because he has several scars on various parts of his body showing the seats of old tuberculous abscesses. Some tuberculin was put into one eye, and the characteristic reaction known as Calmette's reaction was obtained. In this method of trying to find out if a patient has tubercle, two drops of tuberculin in a 0·5 per cent. solution are dropped on to the conjunctiva of one eye, near the inner canthus, and the lids are held apart for two or three seconds. In a few hours, if the patient is a tuberculous subject, the conjunctiva becomes reddened and there is an overflow of tears on to the cheek, in a few more hours the reaction reaches its maximum, and by the end of forty-eight hours it has subsided. The other eye is unaffected, and neither eye is affected if the patient is not a tuberculous subject. No harm follows. Dr. Eyre has had a very large experience and has never seen any. The test is not infallible but it is extremely valuable. Our patient's opsonic index to tubercle was a little below normal. It was not so much reduced for the reduction to be of much weight in itself had it not been that the other signs of tubercle were so striking. It was 0·85. Please remember that you will frequently find, often in the lungs or bones, evidence of tubercle in Addison's disease. Addison himself thought that malignant disease of the supra-renal capsules would cause the disease. Then a wave of opinion sprang up which taught that the change in the supra-renal capsules in Addison's disease must be a caseous tuberculous change; but Addison had a wider conception, and thought that any disease which impaired the functional activity of the supra-renals would cause the disease that bears his name. He believed the supra-renal bodies poured something into the blood, and that anything which destroys them will prevent this something getting into the blood, and hence the disease. And he has turned out to be correct, because we now believe and know that any lesion of the supra-renal will, if sufficiently extensive, produce Addison's disease, whether it is tubercular disease or malignant disease. All his eleven cases were undoubted examples of Addison's disease, yet several, as you have just seen, were not examples of tubercular disease.

The next point I want to draw your attention to is the last specimen, in which the capsules have undergone considerable atrophy. Much discussion has been excited as to the cause of this. One thing is quite certain, that sometimes the condition of those capsules corresponds to fibrosis of the lungs, and we may fairly assume that there has been old tubercular trouble followed by fibrosis, and hence atrophy. In other cases we do not know the cause of the atrophy.

Subsequent experience has shown that the number of cases of malignant disease of the supra-renals causing Addison's disease is fewer than appeared from Addison's eleven cases. Of those, no fewer than three were due to malignant disease. But in subsequent experience it has been found that by far the greater number of the cases of Addison's disease are due to obvious tuberculosis of the supra-renals, and the number is still higher if you include, as certainly sometimes you ought, among tuberculosis the cases in which the glands

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have simply undergone atrophy. You may ask why do so few cases of malignant disease of the supra-renals cause Addison's disease, for it is admitted that there are many cases of malignant disease of the supra-renals in which no Addison's disease is present? I think the reason for that is not far to seek. You must first have both supra-renals affected. And in the next place it may very well be that malignant disease will kill the patient before it destroys the supra-renals, and so produce Addison's disease. There are very few cases on record illustrating Addison's eleventh case, in which, owing to the vein going to the supra-renal being blocked, there was hæmorrhage into the gland, but it appears that acute hæmorhage into the supra-renals may sometimes cause the symptoms of Addison's disease. You see nothing substantially has been added to the description of Addison's disease since his death, and I think that is a great monument to his power of observation. Absolutely the only clinical observation which has been added has been that we have, by a manometer, tested the truth of his statement that the pulse is small and feeble.

Not every case, in which the clinical symptoms are such that it is difficult to resist the diagnosis of Addison's disease, that has ever been published shows disease of supra-renal capsules. As a matter of fact it is stated only 88 per cent. do. But I think that is well within the limits, because some cases may have been recorded as Addison's disease in which the diagnosis was erroneous. Sometimes the post-mortem examination may have been inefficient. And again you have to remember that sometimes there may conceivably be some functional disturbance of the supra-renals. Another thing you have to bear in mind is that we do now know that cells very similar to the cells in the medulla of the supra-renals are found in several situations, for instance in the small inter-carotid glands which we have on either side of the neck, and the coccygeal glands have chromophile cells, so-called because of the ease with which they take up chromic acid like the cells in the supra-renals. And perhaps very rarely disease of these glands may cause Addison's disease and lead to death before the supra-renals are affected. It may possibly be that when disease is found in the supra-renals and yet no Addison's disease has been present, the cells of a similar nature elsewhere in the body have been able to form enough internal secretion to prevent the patient from having Addison's disease. When our patient dies we shall try to dissect out the inter-carotid and coccygeal glands and see if they are atrophied. Perhaps they may be, as the patient is so very ill. Further, it may be that when you find disease of the supra-renal bodies and yet no Addison's disease is present, that the disease of the supra-renals has not been extensive enough to completely destroy the medulla of the organ.

I only want to tell you now one or two points, not clinical, which we have learnt of late. We do know that the cortical part of the supra-renals is distinct from the medullary. As far as we know, disease of the cortical part of the supra-renals has nothing to do with Addison's disease. In the few cases in which tumours have been recorded as growing from the cortical part of the supra-renals, some of the patients have presented abnormality of sexual development, nearly always an extremely precocious sexual development, the genital organs are large, the hair on the pubes comes at an unusually early age. These cases are very rare, and do not interest us this afternoon. The great discovery of the last few years in connection with the medulla of the supra-renal capsules is that it manufactures an internal secretion, and that this secretion maintains the blood-pressure throughout the body, being antagonistic to the depressor secretion of the thyroid gland. This is a discovery which helps very much in the understanding of Addison's disease, because we can now see why, owing to the failure of the body to have enough of this substance, adrenalin as it is called, circulating through the system, the blood-pressure falls lower and lower, until finally it falls to the extremely low point of 48, as Dr. Mann has observed in our patient. And that opens up a very interesting speculation, which is quite worth working at for any of you who want to write a thesis. It has been put forward, and I think with some truth, that possibly some people thought to die from other diseases really die in the early stage of Addison's disease. What I mean is, that supposing a man has malignant disease of some other part of his body, and his pulse becomes feebler and feebler and of lower tension, it is very tempting to assume that what is the matter with him is that his supra-renal bodies are failing to secrete enough adrenalin, and hence his pressure has fallen, and that contributes to his death. At any rate there is no doubt that very often, when the pulse is of very low tension, and very feeble, it can be brought up again in clinical work by injecting some adrenalin into the circulation. We have had to do it twice in clinical ward in the last week, and

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it is striking how quickly the pulse has recovered after that has been done.

The suggestion has often been made that some of the symptoms of Addison's disease, *e.g.* the vomiting, are due to implication of the nerves of the solar plexus in the disease which affects the supra-renals, but it rests on very slender foundation, for in many cases of Addison's disease the semilunar ganglia and nerves have been shown to be quite healthy; they have been found to be affected by malignant disease without Addison's disease being present; and lastly, I know from cutting dozens of sections of the semilunar ganglia that they vary so widely within the limits of health that opinions upon abnormal histological changes in them should be received with great caution.

After the lecture I want you to go into the ward and contrast with Addison's disease the condition of the man in No. 2 bed, John Ward, who has pernicious anæmia, because in the same book that Addison wrote on disease of the supra-renals pernicious anæmia is described. Addison first made the discovery of this disease, and it was while looking about and collecting cases of pernicious anæmia that he at the same time discovered Addison's disease with bronzing. He thought that anæmia was a striking feature of Addison's disease. I think that if you read his account you will see that he did not think it so striking as some other writers have considered he did, and we have learnt of late years that anæmia is not necessarily a striking feature of Addison's disease. In fact our patient in the wards at the present moment has the normal number of red cells, and about 70 per cent. of hæmoglobin. His white cells are slightly in excess. So remember that Addison's disease does not, as a rule, present much anæmia, but idiopathic anæmia, as Addison called pernicious anæmia, which I lectured about not long ago, has very profound anæmia, and, as I said just now, you can see two patients in John Ward, one with genuine Addison's disease, the other with pernicious anæmia.

The treatment of Addison's disease is very unsatisfactory. We gave our patient adrenalin both subcutaneously and by the mouth, but it did no good; in his case it did not even raise his blood-pressure. It has been tried in so many cases without success that when success is reported one cannot help thinking that either the success was not permanent or that there must have been a mistake in diagnosis.

The outlook in Addison's disease is very grave; nearly every patient who has it is dead within two years of the onset of symptoms.

NOTE.—The patient died and both supra-renals were found to be converted into large caseous masses.

March 16th, 1908.

LECTURES

ON

CHRONIC INTESTINAL OBSTRUCTION.

By H. L. BARNARD, M.S.,
Surgeon (with charge of Out-patients) to London Hospital.

LECTURE II.

CARCINOMA OF THE COLON.

GENTLEMEN,—To-day I propose to speak of carcinoma of the colon as a cause of chronic intestinal obstruction. It is second only to intussusception as a cause of intestinal obstruction, and accordingly I find that in the list of fifty-seven cases upon which these lectures are based eighteen were due to this condition. Of these eighteen, thirteen survived colotomy, four died and one refused operation, so that you will see that at least as regards the palliative operation of colotomy the mortality is nothing like so severe as in acute intestinal obstruction, in which anything above 50 per cent. of recoveries is regarded as a surgical triumph.

At the London Hospital between the years 1893 and 1905, 669 cases of all forms of intestinal obstruction were admitted, and of these 151 were due to cancer.

Symptoms.—The clinical picture of the onset of carcinoma of the colon is as follows: An elderly person who has previously had very little abdominal trouble begins to suffer from dyspepsia, flatulence, slight colicky pains and irregularity of his bowels. Very soon he notices that he is losing weight, and that his clothes no longer fit him closely. The flatulence and colic become more marked and exhibit those characteristics as to the side on which they occur and the point at which they stop to which I have already alluded. Perhaps morning diarrhœa makes its appearance if the carcinoma be low down in the colon, and blood and slime are frequently found with the motions, which are often small in calibre, or take the form of small scybala. As the case progresses attacks of acute intestinal obstruction make their appearance. In these attacks the patient suddenly becomes distended. Constipation becomes complete. Vomiting comes

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on, but these attacks are at first relieved by enemata and by purgatives, and are followed by periods of diarrhœa until the condition of alternating constipation and diarrhœa is established. After three or four months of such symptoms peristalting coils of gut may easily be seen through the abdominal wall, and not infrequently the carcinoma itself may be palpated either from the rectum or in some accessible part of the colon. It is very important to remember the common situation for these growths and to appreciate that they become very much more common from the ileo-cæcal valve to the anus. Thus my eighteen cases were distributed as follows: One at the ileo-cæcal valve, three in the ascending colon, four at the splenic flexure, five in the sigmoid, and five in the upper part of the rectum. And this is the customary distribution of a number of cases, so that the balance of probability is very great in favour of a carcinoma being in either the sigmoid flexure or the rectum, and that is why left inguinal colotomy is so often applicable.

I have analysed the distribution of all the cancers of the alimentary canal which are contained in the medical and surgical records of the London Hospital from 1900 to 1905. They amount to 1004 cases, and in the following table I have appended Nothnagel's figures for comparison.

Source.	Œsophagus.	Stomach.	Duodenum.	Jejunum.	Ileum.	Small gut.	Appendix.	Cæcum and ileo-cæcal.	Ascending colon.	Hepatic flexure.	Transverse colon.	Splenic flexure.	Descending colon.	Sigmoid flexure.	Rectum.	Total.
'London. Hospital Med. and Surg. Records,' 1900–1905 . . .	145	378	5	3	2	5	—	41	6	3	17	12	6	103	278	1004
Nothnagel, 'Path. Inst. Wien.,' 1870–1893	—	—	7	—	10	—	2	23	6	—	80	—	—	53	162	343

Pathology.—Carcinoma of the colon is always of the tubular columnar-celled variety. It starts in the submucous tissues and shows a great tendency to spread round the circumference of the intestine as a ring following the lymphatics and vessels. This feature of the growth explains why in so many cases it constricts and contracts the lumen of the gut until it is reduced to an aperture through which in some cases only a probe will pass. This form of carcinoma is frequently very benign in its type. It infects the lymphatic glands, but late, and the liver often does not contain secondary deposits until more than a year has elapsed. As an example of this I may refer to a man on whom I performed colotomy more than two and a half years ago and who refused excision of the growth, and who is still at work, and has only just begun to suffer from cancerous cachexia although his pelvis is now filled by a large mass of carcinoma. The relative infrequency of secondary deposits in cancer of the colon at an early stage renders their excision a most satisfactory operation.

Hansen ('Das Cylinder epithel Carcinom des Magens und des Dickdarms,' Jena, 1890) has studied the occurrence of secondary deposits in the various forms of cancer of the colon. In the scirrhous form of cancer the deposits are chiefly in the liver; the colloid form, on the other hand, has a great tendency to invade the peritoneum and the omentum, whilst the metastases of encephaloid cancer are mainly in the appropriate lymphatic glands.

The clinical division of cases of carcinoma of the colon into those which exhibit symptoms of obstruction and constipation and those which suffer from continuous diarrhœa, is due to the pathological division of these growths into those that constrict and those that ulcerate. I show you here two specimens which illustrate this point. In the first of these, which I excised from an Armenian some four years ago, you will see there is a large ulcer three inches long and three inches wide on the wall of the ascending colon. It is surrounded by a heaped-up edge of carcinoma, and you will readily perceive that it caused barely any real obstruction to the passage of the intestinal contents except in so far as it hardened and indurated the wall and prevented it from peristalting. The other specimen is in marked contrast to this. It was taken from the sigmoid of a woman at an autopsy, in whom symptoms of very acute in-

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testinal obstruction supervened on very slight chronic symptoms, which had persisted for only a short time. You will see that the mass of growth is insignificant, but that it has puckered up the wall of the colon as though a piece of string had been tightly tied around it, and that where the gut is slit open so that the aperture may be seen only a probe is able to pass through.

Whilst, however, from a practical point of view these two forms of cancer of the colon are the most important, many others are found less frequently at operations and autopsies, and a complete list would include the following forms:

(1) The nodular variety.
(2) The polypoid variety.
(3) The annular or ring carcinoma.
(4) The rigid tube.
(5) The papillomatous or cauliflower mass.
(6) A large abdominal tumour.
(7) The gangrenous and ulcerative form.

The secondary changes and results produced by carcinoma of the intestine are also very numerous, and give to the disease its protean clinical forms.

As has already been pointed out the growth in most cases leads to a chronic stenosis of the intestine, but the case is often only seen when it has passed into an acute stage owing to rapid dilatation of the colon or cæcum. We shall return to these complications later.

In rare cases an intussusception may be produced or the colon above the growth may undergo volvulus, bending or kinking.

In many cases a cancer of the colon will produce peritonitis, and this may be either localised around the growth or the whole peritoneum may be infected by a crude perforation or by the transudation of micro-organism through gangrenous patches at the site of stercoral ulcers.

Perforation with infection of the peritoneum may occur in four situations; either through the growth, or in the colon just above it, or in the cæcum when this is suddenly dilated, or an infected and suppurating gland in the mesentery may rupture into the general peritoneal cavity.

Some cancers of the colon establish fistulous communications either with neighbouring coils of intestine or other hollow viscera or through the skin. When such a fistula connects the intestine above and below the growth the symptoms of obstruction may be spontaneously relieved.

Finally the rectum may become ballooned, and this is so curious a phenomenon that a few words on its explanation and relation to cancer of the colon will not be out of place.

Ballooning of the rectum as a result of carcinoma of the colon.—This curious phenomenon is frequently found in other conditions than cancer of the lower colon, such as fæcal accumulations, appendicitis, pelvic tumours, peritonitis, and conditions of neurosis. I have encountered a well-marked instance in a case of hæmorrhagic colitis. The features of this condition are as follows: When the index finger is passed up the anus it appears to issue through a hole in a septum into a large cavity filled with gas. The anal canal is shorter than is natural, and the tonic contraction of the sphincter tighter. When the finger is bent over to explore the interior of the cavity this is found to well-nigh fill the pelvis, displacing the other organs, and to be about the size of a large orange. The mucous membrane is smooth and tense and not velvety and rugose as it should be. On reaching up to the roof of the cavity, in rare cases a cancerous growth may be felt. More frequently the upper part of the rectum is tightly constricted like a sphincter, and may even project into the cavity like an os uteri. The cavity is filled with gas, and on several occasions on which I have introduced a rectal tube this gas was expelled with considerable force and the rectum collapsed. It is stated that ballooning of the rectum disappears under anæsthesia, and probably this is due to relaxation of the sphincter and expulsion of the gas as the patient passes through the period of excitement.

The explanation of this phenomenon has been much debated. Sir Frederick Treves regards it as "due to some phase of paralysis" by which the bowel is dilated and fixed "like a pupil which has been dilated by atropine," but it is difficult to conceive of a segment of the bowel so paralysed that its walls are converted into a rigid shell capable of supporting the intra-abdominal pressure. The passage of a rectal tube which leads to the escape of the gas under pressure negatives this view, and we must conclude that the rectum is distended by flatus. It would appear that the irritation of the adjacent cancer produces spasm of the sphincter and corresponding relaxation of the detrusor of the rectum. Flatus, therefore, collects and distends

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the relaxed part of the rectum, which is inhibited from expelling it through the unduly tight sphincter. It is, moreover, probable that the stricture acts as a strainer in some cases, retaining the solid and fluid fæces but permitting flatus to pass freely.

The increased tone of the sphincter ani will also explain the small calibre of the fæces, which are undoubtedly seen in some cases of colonic cancer. It is impossible that this small calibre can be impressed upon the fæces by a stricture some distance up the colon and be retained during their further passage and expulsion from the anus. Moreover, the intestinal contents are usually fluid above a stricture and are passed through it in that condition, only to become formed when the stenosis is passed.

Treatment.—The treatment of cases of carcinoma of the colon depends upon the stage at which the patient is seen. If the symptoms of obstruction are well marked, our first care must be to relieve these at once by opening the colon above the cancer. But if the case is of the ulcerating, only slightly obstructing variety, and the growth is so fixed as to be inoperable, I believe it is better to postpone the colotomy as long as possible, although this is a debated point. A patient who has had colotomy performed is always more or less incapacitated from following his employment. The very presence of the colotomy opening exercises a depressing influence upon his mind, and whilst I have known several patients of the lower classes who have lived happily with a colotomy wound for a year or more, yet I think that the advantage to be gained by diverting the fæcal stream from the malignant growth is not proportionate to the pain, inconvenience, and annoyance which follow this operation. How, then, can we postpone colotomy for the greatest length of time with the least detriment to the patient. Our first attention must be to the food, and this must be composed of things which can easily be digested and absorbed, and must be free from vegetable matter, which will add to the mass of the fæces. The teeth must be attended to so that the patient may thoroughly masticate his food. In poor patients, many of whom are nearly edentulous and cannot afford a complete set of teeth, I have found it a good practice to recommend them to buy a sausage machine and to pass everything they eat through it. One is then certain that the food is properly divided before it is swallowed. We should next direct our attention to what may be called the sanitary condition of the alimentary canal. By means of laxatives the gut should be emptied completely at least once a week, so that none of its contents may stagnate and decompose above and around the growth. We may then use certain antiseptics in order still further to prevent this decomposition, namely, calomel gr. $\frac{1}{10}$, beta-naphthol gr. ij, salol gr. v, or creosotal gr. x or xv.

When the case of carcinoma of the colon is showing definite obstructive signs we should not wait for these to become more definite and acute but should at once perform laparotomy, determine the situation, extent and fixity of the growth, insert a colotomy tube above it, and at a subsequent operation excise the growth. It may be definitely stated that the surgeon should never attempt to remove the growth when any marked degree of intestinal obstruction is present, but that he should first relieve the obstruction, and ten days or a fortnight later remove the growth. For in the first place the patient's condition is usually so bad that as little surgical interference should be carried out as possible, and in the second place it is a difficult matter to suture the enormously distended colon above the stricture to the small contracted colon below it. The matter is often made still more difficult by the presence of a collection of fluid fæces above the growth. The operation for the exploration and treatment of a chronic intestinal obstruction due to carcinoma should always, I believe, consist of, first of all, a median laparotomy to determine the situation and extent of the growth, and the presence or absence of secondary deposits in the liver and lymphatics, and then of a colotomy suitably placed to relieve the obstruction. Until the abdomen is opened one can never be certain where the growth is placed unless it can be definitely felt in the rectum or through the abdominal wall. When the peritoneum is opened the cæcum is first examined, and if it is distended we know that we are right in our diagnosis of obstruction of the colon. One next examines the sigmoid flexure, and if this is distended it is clear that the obstruction is between it and the rectum, and this may readily be felt from the median incision, and its extent and the presence or absence of adhesions to the bladder or iliac vessels may be ascertained in order to determine the method and advisability of a future excision. One can then cut through the abdominal wall in the left iliac region, making the smallest possible aperture through which the dilated sigmoid flexure can be passed, and, moreover, with one hand inside

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the abdomen and another out there is no difficulty in settling for certain that the coil of sigmoid brought through the abdominal wall is not twisted, and a Paul's tube can therefore be passed with confidence up into the descending colon. I usually fix the coil of sigmoid with a pointed ebonite rod passed through the meso-sigmoid and then close the median abdominal wound and apply dressings. I seldom use stitches to attach the coil of gut to the parietal peritoneum, but simply wind iodoform gauze round and round the coil of gut, passing it beneath the ends of the ebonite rod. This effectually prevents any fæcal matter escaping or leaking into the peritoneal cavity. I then pass stout silk through all the layers of the gut in a purse-string suture and tie the ends with a surgeon's knot which is not yet pulled tight. A Keith's tube of suitable calibre with a long rubber attachment is next laid ready, and an incision is then made with a pair of scissors through all the layers of the gut wall within the purse-string suture. The end of the Paul's tube is rapidly inserted, and by pulling down the ebonite rod towards the lower angle of the wound it is easy to slide the Paul's tube through the gap in the abdominal wall so that its end lies in the descending colon. The purse-string suture is then pulled tight. I think it important that the glass tube should be passed through the abdominal wall, for its presence squeezes the sides of the gut tightly against the wound, and not only prevents fæcal matter from leaking in from without but also the escape of small intestine by the side of the great gut—a very deplorable accident. The Paul's tube is usually left in four days, by which time fæcal matter is usually escaping around it and its utility is passed. The ebonite rod is not removed for ten days or a fortnight so that an efficient spur is formed, and if it is wished the projecting loop of intestine may then be cut off without an anæsthetic, but I do not usually find this necessary. An india-rubber colotomy pad should now be fitted, and personally I prefer the one with an india-rubber plug, for this appears to me not only to prevent the escape of fæcal matter but also prolapse of the gut, and where the plug is not used there is a great tendency for the aperture to close up, and it is often painful and difficult to re-dilate it.

When the sigmoid is found collapsed in exploring a case of chronic intestinal obstruction it is clear that the obstruction is somewhere between the sigmoid and the cæcum. We must then enlarge our incision at least as high as the umbilicus, and by pulling on the omentum the transverse colon may easily be brought into the wound. If this is distended then the obstruction is almost certainly at the splenic flexure, where it may be felt if we run our hand up the transverse colon to it, or it may lie in the upper part of the descending colon over the front of the left kidney. Under these circumstances I believe the right treatment is to fix the transverse colon in the upper angle of the wound and to open it in the manner I have already described. Some surgeons seem to have a great objection to transverse colotomy, but in my experience every inch of the colon which intervenes between the ileo-cæcal valve and the colotomy wound is of value in saving the patient from an irritating eczematous aperture. By astringents and antiseptics a patient with transverse colotomy and using a plug colotomy pad suffers very little more inconvenience than if his colotomy were in the sigmoid. Where the growth is at the hepatic flexure we must of necessity open the cæcum, but this I am convinced is an operation which should be avoided as much as possible, for the fluid, acrid, digestive contents of the small gut excoriate, inflame and ulcerate the margins of the wound, and it is very difficult to find any contrivance which prevents them from escaping into the dressings.

Where the preliminary investigation of the growth has indicated that it is one suitable for excision this procedure should be carried out ten days or a fortnight after the obstruction has been relieved. Unfortunately in the great majority of cases in my experience no operation is possible. For there are either extensive secondary deposits in the mesenteric glands, or the liver, or the growth itself has already invaded the bladder or is fixed on to the abdominal wall or to the iliac vessels. Indeed, of eighteen cases I was only able to perform excision in two. It is therefore very important that the general medical profession should become much better aquainted with the early symptoms of carcinoma of the colon, so that they may consult the surgeon in the early stage of the disease, when it is small, localised, and without adhesions to important structures. And that was my reason for speaking at such length upon the symptoms of this condition. Sometimes at the time when one has opened the abdomen for the intestinal obstruction it is found that the carcinoma is freely movable and may be brought out of a suitable abdominal incision, and when possible this should always be done, as the growth may then be shaved off after a few days without any risk of infecting the peritoneal cavity. And it only remains then to close the colotomy wound. But where the growth cannot be pulled outside the abdomen it is necessary at the second operation to re-open the peritoneum, remove the growth, and suture the ends of the intestine together. As Caird, of Edinburgh, has so ably urged, suture is not much slower than Murphy's button and the result is very much more reliable. I show you here two specimens of portions of the colon that I have removed for carcinomatous growth, and I am glad to say that both these cases survived and did well. The

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first consists of two inches of the ileum, the cæcum, appendix, ascending colon, half the transverse colon, and a large mass of glands which were situated by the side of the ascending colon. This mass of intestine was removed in one piece and laid bare the psoas muscle and the lumbar plexus, the right kidney and ureter, the second part of the duodenum, and the side of the vena cava. The end of the transverse colon was closed by suture and the termination of the ileum was grafted into the side of it. It was necessary to drain the enormous area of cellular tissue uncovered by the peritoneum, but after a fortnight's suppuration the wound closed up and the man left the hospital one month after the operation. This was four years ago and no relapse has occurred. The other specimen is a localised carcinoma springing from the transverse colon only a short distance from the splenic flexure. I removed, as you see, about four inches and sutured the ends of the gut together. The man made a good recovery and the growth has not recurred after three and a half years, during which time he has been actively engaged in his work. In a more recent case I removed the whole of the sigmoid flexure and rectum with their mesentery and glands by means of the abdominal perineal operation in a man, æt. 72 years, and he also is perfectly well.

March 16th, 1908.

NERVOUS PHENOMENA IN PNEUMONIA.*

By SAMUEL WEST, M.D., F.R.C.P.

HARDLY any case of pneumonia runs its course without developing nervous symptoms of some kind, and I would here premise that pneumonia as used in this address means inflammation of the lung due to the pneumococcus. For the most part they arise during the acute or active stage and pass off rapidly so soon as the acute stage is past. Of those that persist, some, at any rate, have little more than an accidental association with pneumonia, and others stand in rather a distinct relation to it.

Of those which arise during the acute stage, some depend upon the sudden change in physiological conditions which the body undergoes owing to the abrupt onset of the disease. Of this the best instance is the rigor, and another is the general irritation of the brain and spinal cord, which may be so marked as at first to suggest even cerebro-spinal meningitis.

Other of these symptoms may depend upon high temperature, though hyperpyrexia in the strict sense of the term is rare.

* Delivered before the Medical Society of London.

Others again, and this the largest number, probably depend upon the toxines produced. The toxines are many and various. Some may be developed within the bacilli themselves (intra-bacillary toxines); others may be derived from the tissues of the body by the destructive action of the bacilli upon them. Of the many toxines thus produced, some may act especially on the heart, some on the liver or kidneys, some on the nervous system. Under ordinary circumstances a sort of relation is observed between them all, but under special circumstances one or other of them may predominate and give rise to special physiological or pathological effects. In this way may be explained the general family resemblance among all cases of pneumonia as well as the peculiar symptoms which give character to each individual case. These individual peculiarities, whatever explanation we give of them, it is most important to study, for upon their adequate recognition in any given case successful treatment largely depends. With this short preface I pass to the nervous phenomena to which I propose to refer.

(1) *The Nervous Phenomena of the Acute Stage.*

The rigor.—Shivering is rarely absent in some degree as an initial symptom, and in about 80 per cent. of all cases takes the form of a very severe rigor, which may last twenty minutes to half an hour. There are few diseases in which the initial rigor is so marked or so prolonged. Usually the rigor, if it has been severe, is single, and is not repeated during the attack. It may, however, now and then happen that there are two or three well-marked rigors, but under any circumstance they occur during the first few hours only. Sometimes the place of the rigor is taken by slight shiverings; if so they may be repeated and last throughout the first or even the second day. This only occurs when the pneumonia is of gradual or insidious onset—for, as a general rule, it may be stated that cases of acute onset have a severe rigor. In little children the place of the rigor may be taken by a convulsive fit, as in other acute fevers, but this is much rarer than one would gather from books. Where the fits recur and the child remains unconscious the diagnosis between pneumonia and meningitis may at the time be difficult, but even from the first the rapidity of respiration and the perverted pulse-respiration ratio is usually sufficient to prevent error.

Headache.—Severe frontal headache is one of the most constant initial symptoms, as in other fevers. As the disease develops it usually becomes less severe, or, at any rate, is less complained of. It may, however, be a distressing symptom throughout, especially where, in other respects, the patient is not very ill.

Insomnia.—The sleep is always more or less disturbed and broken. The patients usually doze

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or sleep in short snatches, as the pain, cough, and dyspnœa permit them.

Now and then it happens that without obvious cause the patient cannot close the eyes, and lies awake hour after hour, weary and longing for sleep, but unable to get it. This is a condition of gravity and indicates great nervous excitability, which will be followed by grave depression, and not improbably end in collapse and death. Undesirable as it may be in general to employ opiates or strong hypnotics in pneumonia, still, sleep must be obtained in some way, and it will be wise in such a case to choose the lesser of two evils, viz. to run the risk there may possibly be with narcotics and procure sleep.

This is the place to speak of hiccough. This is fortunately a rare phenomenon, but one of great gravity. For it not only causes great discomfort and pain, but prevents sleep. The hiccough may occur every few minutes or even several times in the minute, and may last hours. I have seen it occur two or three times a minute for a day and a half, and only cease when the patient was moribund. It is most difficult to control, even with full doses of opiate.

Delirium is very common. Few cases of any severity run their course to the end without it. It varies much in degree and kind, and seems to be nearly twice as frequent in apex as in base pneumonia. It may be due to the fever or some poisonous substance generated by it, to hyperpyrexia, to acute complications, of which pericarditis is the most important, to serious changes in the lung *e. g.* gangrene or suppuration, to debility, and lastly, but very commonly, to previous alcoholism. It is usually a prominent symptom in the young and in the aged. In mild cases it may not amount to more than a little wandering or lightheadedness at night; in other cases it persists throughout the day and is aggravated at night, as is especially the case in the aged and feeble. In asthenic cases the delirium is of a low wandering character, with mild hallucinations, the patients lying only half asleep with the eyes half closed, picking constantly at the bed-clothes or at something in the air.

In very severe cases the patients may from the beginning be in a state of more or less stupor and be almost comatose, or they may pass into that condition from any of the other states of delirium.

In another group of cases the delirium may be of a much more active character, and be like that of delirium tremens. This is rare except in drinkers, but may be met with even in very temperate persons. In drinkers delirium tremens develops with all its active and lively hallucinations, and cannot be distinguished from the ordinary delirium tremens except by the fever. Such cases are almost invariably fatal, but the delirium stands in no sort of relation to the extent of the consolidation of the lungs, being sometimes extreme and running on to a fatal issue, when the consolidation is too small to yield physical signs. The delirium is sometimes extremely violent, and it has not a few times been diagnosed and treated as acute mania.

The delirium may change its character, and thus the most active forms pass as the case grows worse into the low wandering asthenic form, and the patient dies at last almost comatose.

Wandering at night may sometimes develop for the first time, or having disappeared may return again, during convalescence. This is generally due to debility, and is cured at once by stimulants.

Children, old persons, and drinkers are usually delirious from the beginning. In all cases alike gradually and steadily increasing delirium is of bad prognosis.

The knee-jerk is normal for the first day or two, disappears on the third or fourth day, remains absent till the ninth, and after being slightly increased for a day or two returns to the normal at the end of the second week. The knee-jerk stands in no relation to the crisis. It is of some value in prognosis, for where it disappears before the third day the prospect is grave, and where it remains normal till the seventh day recovery is practically certain. It may have some value in prognosis, for it appears to be increased in tubercular pneumonia, and to be unaffected in septic or non-pneumococcal pneumonia.*

Subsultus tendinum is only met with in severe asthenic cases, in whom also *muscular tremors* are common. These tremors affect any part of the body which is put in motion, *e. g.* the hands and arms, the lips and the tongue. They are absent during sleep and when the parts are at rest, and are not necessarily associated with delirium. Similar tremors occur in the alcoholic cases, but in them depend upon the drink.

Acute tympanites, or extreme flatulent distension of the intestines, deserves mention here, for it is neuro-muscular in origin and indicates the greatest nervous prostration. It is really a paralytic phenomenon, the muscular tone of the intestines being completely lost; the distension which is the result leads to much pain from the stretching of the abdominal muscles, and to great aggravation of the dyspnœa from the pushing upwards of the diaphragm into the thorax. It is a very serious symptom, extremely difficult to relieve, and almost invariably of fatal omen. The distended intestines may be tapped with a fine trocar without risk, and

* For these interesting observations we are indebted to Dr. Stanley Barnes ('Birmingham Med. Rev.,' April, 1906). The absence of knee-jerks in pneumonia was, I believe, first described by Hughlings Jackson in 1894 ('Lancet,' December 22nd). It was subsequently recorded by Pfaundler ('Münch. med. Woch.,' July 22nd, 1904) as common in children, and he suggested its diagnostic value, as differentiating between croupous pneumonia and early meningitis.

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the gas allowed to escape, but the relief is only slight and transient.

General cutaneous hyperæsthesia sometimes develops, but is of no special importance. It appears early, and rarely lasts more than the first day or two.

Local hyperæsthesia limited to some part of the side affected is not so rare. It is no doubt due to the irritation of the intercostal nerves, which is referred to their peripheral distribution. If it is severe and not relieved by poultices or counter-irritation, a little tincture of aconite brushed freely over the painful area will cure it rapidly.

Spinal irritation.—Sometimes the attack is ushered in with signs which suggest acute spinal irritation, so that spinal meningitis might be thought of, and when these signs are associated with head symptoms, too, acute cerebro-spinal meningitis may be closely simulated. The pulse-respiration ratio is usually sufficient to determine the diagnosis, and doubt, if there be any, lasts only for the first few hours, or at the most a day or two, for with the development of the local lesion these signs of general irritation subside.

Thus there may be stiffness of the back and neck, even with retraction of the head and arching of the back.

Movements may evoke or aggravate these symptoms and cause pain. There may also be tremors or slight spasms of the limbs.

As the cases which simulate cerebral meningitis are most common in children, so I think the spinal cases are more common in the adult; but such may occur in either young or old.

The following case is an illustration: A lad, æt. 14 years, came under observation on the second day of pneumonia. The attack appeared a slight one, and the physical signs were not at first very definite. The temperature was 103° F., the pulse 120, and the respiration 40. He lay straight, somewhat stiffly, on his back, but as soon as he was disturbed in any way coarse tremors appeared in the parts moved, and if he were turned on to the side became general, while at the same time the legs became stiff, the head retracted, and the back slightly arched. The condition suggested cerebro-spinal meningitis, but in twenty-four hours these symptoms passed away, did not return, and the case ran otherwise its ordinary course to recovery.

It is interesting to note that in the same paper of 1894 in which Hughlings Jackson described the altered knee-jerks he also, from certain respiratory-muscle symptoms, suggested that the pneumo-toxines, as the toxines of pneumonia, not infrequently produced some pathological change in the motor cells of the cord.

Transient paralyses, e.g. aphasia, facial palsy, even hemiplegia or monoplegia are described. The attacks develop early, last only a few hours, occasionally for three or four days, but end almost invariably in complete recovery. They are probably, for the most part, toxic in origin. Similarly, transient disturbances of vision, photophobia, and occasionally amblyopia have also been recorded. These are all very rare complications in this country. Most of the instances are recorded in French literature.*

(2) *The Nervous Phenomena which Persist after the Acute Stage.*

These might be called sequelæ, but some of them originate during the acute stage.

Meningitis is extremely rare; it did not occur once in either of two series of 100 cases recorded by me. Osler met with it, however, in eight out of 105 cases. This is as unusual as his experience of acute endocarditis, and as five out of the eight cases were associated with that affection it is probable that these, at any rate, were embolic.

The inflammation appears to be always purulent, and in the pus the pneumococcus has been found alone in some cases; in others associated with other organisms; in still other cases the pneumococcus has been absent and streptococci or other organisms present, alone or in association. Meningitis associated with the pneumococcus has, of course, been found without pneumonia.

In many of the cases the meningitis has been found on post-mortem examination, without having been suspected during life, but when symptoms are produced they are of the ordinary kind.

Epidemic cerebro-spinal meningitis is frequently associated with pneumonia (nine times in thirty cases, Immerman), and in a considerable number of cases in which there has been no pneumonia the pneumococcus has been found in the pus. I think it would be safer to say an organism like the pneumococcus, and difficult to distinguish from it rather than to assume that it is the pneumococcus itself, for it is difficult to accept the view that the same organism can be the cause of two diseases so different in themselves and so rarely associated, except when cerebro-spinal meningitis is epidemic

Hemiplegia, when not transient, and therefore probably of toxic origin, must be due to some local lesion—thrombosis or embolism.

In young persons the attack may be due, as in specific fevers, to a local infective endarteritis; in elderly persons to some atheromatous or other sclerotic condition of the vessel, which alone or in association with infection has led to thrombosis. Hemiplegia due to hæmorrhage can hardly stand in any but an accidental relation to pneumonia.

Pneumonia, like other severe illnesses, is sometimes followed by grave mental symptoms, *e.g. mania, melancholia*, or *imbecility*. As a rule the

* Chantimesse, 'Bull. et Mem. de la Soc. Med.,' December, 1893.

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mental functions are in the end restored, though convalescence may require months of care and watching.

When the mental degrees persist cerebral softening and degeneration may be found. This is generally due to defective nutrition through stenosed vessels, but may be of toxic origin.

Peripheral neuritis has been recorded. It develops during convalescence and is in all respects like that which follows diphtheria, for it is toxic in origin and ends in complete recovery.

If the affection of the knee-jerks in the acute stage is due to a peripheral nerve lesion, it is strange that peripheral neuritis is so rare after pneumonia as it is.

I do not propose to enter into the more difficult theoretical question of the part the nervous system plays in the acceleration of respiration and the disordered action of the heart. The local conditions in the lung appear sufficient, in most cases at any rate, to explain the rapid breathing, while the direct effects of toxines upon the heart muscle seem adequate to account for the irritability and weakness of the heart. It is evident that the nervous system plays a more important part in pneumonia than is ordinarily supposed.

The PRESIDENT (Dr. KINGSTON FOWLER) thought physicians had yet to learn the clinical aspects of the different forms of pneumonia. He referred to the subject to his students lately in connection with a case which was transferred from the gynæcological wards of Middlesex Hospital to one of his own wards, having developed pneumonia four days after delivery outside the hospital. He could not help being struck by the absence of many of the characteristic features of pneumococcal infection. He said, at a previous discussion at the Medical Society, that many of the symptoms not locally connected with the lung, such as dyspnœa, in pneumonia were symptoms of the pneumococcal infection. In that particular patient, although there was complete consolidation of both lobes of the left lung, there was complete absence of many of the symptoms ordinarily associated with pneumonia, and that led him to say that no student would be expected to say that that was a case of pneumonia, although the patient was in the seventh or eighth day and had a temperature of 104° F. Her facial appearance was absolutely different from that of a case of pneumococcal infection. Pneumococci were absent from the sputum, but streptococci were present. Therefore other organisms could cause consolidation. Not only were there varieties of virulence, but clinical varieties of the illness had to be recognised as due to different organisms. He agreed as to the importance of sleep, and the necessity of avoiding the giving of narcotics. Hypodermic injections of morphia in the later stages of pneumonia were very dangerous, but they might be useful. Hiccough was a rare complication, but he remembered a case in which it occurred and lasted four or five days. He reported the case at the time. It was relieved by hypodermic injections of morphia. He thought it would end fatally, but it did not, and he thought the determining factor in the recovery was the morphia. Delirium often lasted a considerable time after the crisis in pneumonia. With regard to stimulants, the longer experience he had the less he was inclined to give alcohol in the disease. He had a case in which six ounces of brandy per day had been given before admission, but when the patient came to his wards he discontinued that, and there was no traceable ill-effect from it; in fact he believed the patient was better. He agreed with Dr. West that paralyses and other symptoms of spinal irritation must be very rare. Peripheral neuritis was also rare, but he had seen it following incomplete resolution of basal pneumonia.

Dr. DE HAVILLAND HALL pointed out that the head symptoms in pneumonia were influenced by the position of the pneumonia. Head symptoms were most pronounced when the pneumonia was apical. That was so much the case in children that in the wards it was his habit to point out that before arriving at a diagnosis of meningitis in them, the possibility of pneumonia being present should be carefully excluded. Sometimes apical pneumonia was central, and the symptoms appeared twenty-four to forty-eight hours before there were any physical signs. Not long ago he accepted for admission to hospital a case by a former house-physician, a careful man, which he had sent as one of tuberculous meningitis. He pointed out to the students that it was important to exclude pneumonia, and upon percussing over the left apex a dull note was heard and distinctly tubular breathing. It turned out to be a case of typical apical pneumonia. One occasionally saw true meningitis in connection with pneumonia, and there was Dr. Osler's series of cases of meningitis and pericarditis. He (Dr. Hall) thought both those serous surfaces were often affected at the same time. In a doubtful case of meningitis it was well to have lumbar puncture performed and examine the fluid, because in many such cases the pneumococcus had been found. Where the patient was maniacal in the early stage and jumped out of bed and ran round the room, the form of the disease was usually severe. He said he recently had a case of hiccough in connection with a severe case of suppuration of the gall bladder, and there five grains of chloretone every four hours afforded great relief, everything else having failed. Pilocarpine was also occasionally useful. It had been his own practice also to give less and less alcohol. It was always gratifying to him, when he had a severe case of pneumonia in the ward, to be able to

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demonstrate to the students that it could get well without stimulants. If he did use stimulants at all he reserved them for the time of the crisis.

Dr. E. CAUTLEY said he knew it was a recognised text-book theory that rigors in adults gave place to convulsions in children suffering from pneumonia, and he asked whether that was Dr. West's experience. In his own experience it was unusual for true pneumonia to have been ushered in by convulsions in children. The commonest initial symptom in them was severe vomiting. He did not think meningitis was often met with in children having pneumonia, but meningitic symptoms were found in them. Occasionally there was a true pneumococcal meningitis, which had no lung symptoms, ending in a crisis, and verified during life by examination of the fluid obtained by lumbar puncture. Usually those cases were fatal, and were secondary to pneumonia. Nervous symptoms in connection with the disease were not very common in children. Where nervous symptoms were present it was necessary to be sure that the disease was pneumonia and not influenza, in which nervous symptoms were frequent. In children the mortality from influenza was very low, and where a child had pure pneumonia one could almost prophecy it would recover. Alcohol was rarely necessary in children, and only asthenic cases required it, whether adults or juveniles.

Dr. F. J. POYNTON asked how it would be best to treat a man, æt. 23 years, in about the third day of pneumonia, who was actively maniacal and unable to sleep. With regard to meningitis in association with pneumonia as the result of pneumococcus, he recently analysed 100 consecutive cases of suppurative pericarditis, the majority of which were pneumococcal. In eleven of them there was suppurative meningitis as well, and in several of those the pneumococcus was demonstrated in the meninges and in the fluid round the pericardium. In very young children there seemed to be two chief types: (1) A primary type coming on almost with the pneumonia, and that was very rare; (2) the marasmic form, with very few symptoms, but unexpectedly proving fatal. He supposed that where the curious nerve symptoms developed late in the pneumonia it was a central pneumonia, but it was also possible that those symptoms were due to pneumococcal infection, more or less general, and that at last it appeared as a patch of pneumonia, not necessarily coming from the depths. Sometimes there was a dropping of the pulse to 40 or 50, which was probably a vagus effect, this eventually disappearing. He thought there might be a primary pneumococcal abscess of the brain. In two patients with severe pneumonia the illness was complicated by measles, and there suddenly appeared severe cerebral symptoms. It was thought to be meningitis. Finally it quieted down, leaving a right hemiplegia, and in both instances that all remained permanent. In his experience it was very exceptional for convulsions to replace rigors in children; rigors seemed to be almost the private property of the adult.

Dr. WEST, in reply, said he had assumed the definition of pneumonia had been understood, and his remarks were based upon that assumption. He agreed with everything that had been said in regard to stimulants. The condition of the patient at the time when large quantities of alcohol were given was, he thought, worse than now, and stimulants were only exceptionally necessary, and that was in asthenic cases, whether young or old, and where there was danger of collapse at the crisis. It was not uncommon to find the delirium lasting for some time after the crisis. And the stimulant need not always be alcohol; strychnine could be used. He did not think it harmful to give opiates in pneumonia where the disease was localised and the rest of the lung was free. Where the lungs were greatly congested and there were bronchitic signs, opiates were best avoided. He had, he thought, pointed out, that head symptoms were worst in cases of apical pneumonia. The difficulty of diagnosis in the case of children was not so great as the books led one to believe, for in nearly all the grave cases the pulse-respiration ratio was a guide. In meningitis there was not the same acute onset or the perverted pulse-respiration ratio which was found in pneumonia. In speaking of rigors being replaced in children by convulsions he offered no idea as to their frequency or rarity. In answer to Dr. Poynton he assumed that that gentleman regarded pneumonia as a general blood infection, with local manifestations, whereas he did not do so. If the pneumococcus was always the same thing there was much to be said for Dr. Poynton's view. He knew the pneumococcus had been found much more frequently in the blood than was anticipated, but it possibly was not always the same microbe. He believed that pneumonia was a definite clinical entity with a series of complications, and was different from a large number of the cases called pneumococcal infection. Slowing of the pulse and irregularity of the heart were quite common, and it was sometimes a week or two before they recovered. The hemiplegia after pneumonia seemed of the same kind as that after such a disease as measles, where during an attack of measles infective endarteritis occurred. That was not limited to vessels of the brain, but was found in other parts of the body. He remembered a case of peripheral gangrene in which all the fingers dropped off, and that was due to local arteritis of some branches of the arteries in the hands. There had been many cases of hemiplegia after septic disease in children, which had been found to depend on a similar infective arteritis in the vessels of the brain.

March 16th, 1908.

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A LECTURE

ON

PNEUMONIA IN CHILDHOOD.

Delivered at the North-East London Post-Graduate College.

By T. R. C. WHIPHAM, M.A., M.D.Oxon., M.R.C.P.Lond.,

Physician to the Evelina Hospital for Children; Assistant Physician to the Prince of Wales' Hospital.

PNEUMONIA in childhood is a subject about which in many minds a good deal of misapprehension still seems to exist. It is generally taught that broncho-pneumonia is most commonly found in infancy and old age, a fact which I do not dispute, but the statement is apt to lead one to suppose that the lobular form is that variety of pneumonia which is especially associated with early life. This is a point of which I wish in the first place to disabuse your minds, supposing that any of you still labour under this idea. Broncho-pneumonia is certainly the commoner form in infancy, but as compared with lobar peumonia it is not so much more frequent as is often supposed. I have recently looked through my case-books and have abstracted the details of 150 cases of pneumonia occurring in children under twelve years of age, which have been under my care during the last few years. Of these I find that 65, or 43·3 per cent., were of the lobar type, and 85, or 56·7 per cent., were broncho-pneumonia—an approximation which is worthy of your consideration. The differentiation between lobar and broncho-pneumonia is, as you are well aware, often a matter of no little difficulty, and this is a point which I shall touch upon later. As I have said, 43·3 per cent. of the cases were lobar and 56·7 per cent. were broncho pneumonia, or a ratio of roughly 1 to 1½. These figures closely correspond to those of cases at the Shadwell Hospital, given by Pearson, who collected 121 cases of lobar pneumonia and 172 of broncho-

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pneumonia, or 41·3 and 58·7 per cent. respectively, and also with those quoted by F. Taylor* of the cases occurring at Guy's Hospital during the year 1906, of which 28 were lobar and 35 broncho-pneumonia, a percentage of 44·4 and 55·5. The similarity of the figures is striking, and shows that the incidence of lobar pneumonia in childhood is greater than is usually considered to be the case. In contra-distinction to the figures which I have quoted, Holt,† in his American statistics, finds that broncho-pneumonia is more common than lobar pneumonia in the proportion of 3 to 1, and some English writers agree with him in fixing the ratio at a higher figure than I have done.

The division of pneumonia into lobar and broncho-pneumonia is an anatomical one, but the two conditions differ in their clinical manifestations, in their pathological details, and to a certain extent also in their bacteriology. Without further preamble, therefore, I shall proceed to a consideration of each of the two forms separately.

Lobar Pneumonia.

Sex.—In adult life lobar pneumonia, as is well known, chiefly affects males, and the same is true of childhood. In my series, out of 65 cases 37 were males and 28 females, or 56·9 per cent. as compared with 43·1 per cent. This closely tallies with Holt's figures of 60 and 40 per cent., and seems to be the usual ratio. Why the male sex should be especially susceptible to pneumonia, at all events in childhood, is, as far as I know, as yet unexplained.

Age.—Lobar pneumonia may occur at any age. The youngest in my series was seven months, but Holt records a case in an infant at the age of three months, and states that it may be found even in the newly born. The greatest number of my cases occurred during the second, third and fourth years, 37, or nearly 57 per cent., of the cases occurring during this period, as is shown by the following table:

Age.	Cases.	Deaths.
1st year	4	2 (50 per cent.)
2nd ,,	12	4 (33·3 ,, ,,)
3rd ,,	15	3 (20 ,, ,,)
4th ,,	10	1 (10 ,, ,,)
5th ,,	6	—
6th year	5	—
7th ,,	3	—
8th ,,	3	—
9th ,,	1	—
10th ,,	3	1 (33·3 ,, ,,)
11th ,,	2	—
12th ,,	1	—

This coincides with Rivière's observations, but Holt, in dealing with American cases, finds the period of greatest incidence to be from the third to the fifth years, and other authors make it even later still.

Ætiology.—The exciting cause of lobar pneumonia is the pneumococcus, which is present in the sputum of nearly all cases in which it is examined. Children, as a rule, do not expectorate, the sputum that is brought up being generally swallowed. A useful method of obtaining some for the purpose of examination is to make the child cough by touching the wall of the pharynx with a tongue-depressor and then collecting the mucus on a swab as it appears at the back of the throat. The pneumococcus, you will remember, is a small oval-shaped organism, about 1 μ in its largest diameter, generally arranged in pairs or in short chains of from four to ten. It is surrounded by a very definite capsule, and retains the stain in Gram's method. Among the predisposing causes of the disease must be reckoned exposure to cold, and to this is attributable the prevalence of the disease in the early spring months when great or sudden variations in temperature are likely to occur in the course of the twenty-four hours. Occasionally there is a history of a previous attack of pneumonia, and in some instances it is preceded by an attack of measles, whooping-cough, typhoid or scarlet fever. In by far the greater number of cases, however, its onset is primary.

Pathology.—In my cases the right lung was affected in 56·9 per cent. of the cases, and the left in 35·3 per cent., the various situations being:

Right apex	16 cases.
,, middle lobe	4 ,,
,, lower lobe	16 ,,
Whole of right lung	1 ,,
Left apex	0 ,,
,, lower lobe	22 ,,
Whole of left lung	1 ,,

* F. Taylor, 'Wightman Lecture,' 1907.

† Holt, 'Diseases of Infancy and Childhood.'

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Both apices.	2 cases.
„ lower lobes . . .	1 „
Whole of both lungs . . .	2 „

It is generally acknowledged that the right lung is more often affected than the left, but the figures which I have given you show rather a large excess. Thus Holt in a series of 950 cases found the right lung involved in 45·5 per cent. and the left in 41·5 per cent. You will notice that in my cases, where one apex was affected, it was the right which was attacked in every instance, and that in contra-distinction to this the basal cases were found chiefly on the left side. The relative susceptibility of the right apex and the left lower lobe together with the relative immunity of the right middle lobe is further confirmed by Holt's statistics.

As regards the post-mortem findings in cases of pneumonia, I would remind you that three stages are to be recognised, first the stage of congestion, then the stage of red hepatisation, and lastly that of grey hepatisation. Macroscopically the area of congestion is dark in colour, heavy and œdematous. The area of red hepatisation is of a dark red colour and uniform in appearance; it is raised above the surrounding lung tissue, and its cut surface presents a somewhat granular aspect; it is also distinctly friable and airless, and so heavy that it sinks in water. I wish to point out, however, that in children the pneumonic area is, as a rule, less well defined than in the adult, and less apparent to the eye, while its cut surface is more œdematous and does not appear so obviously granular. The further stage of grey hepatisation is characterised by its mottled grey colour, and the extreme friability of the diseased tissue; the lung substance is more moist, and on section has lost its granular appearance. Histologically the congestive stage is evidenced by the distension of the smaller vessels and the exudation of serum and epithelial cells into the alveoli, with swelling of those cells that still remain to form a lining. In the second stage, in addition to the congestion of the blood-vessels, the air-vesicles are found to be filled with fibrin, in the meshes of which are numerous red blood-corpuscles, a few leucocytes and desquamated epithelial cells. The alveolar septa may show a slight degree of small round-cell infiltration, and evidence of a mild bronchitis may be present in the smaller tubes in the neighbourhood. In the final stage, or that of grey hepatisation, the fibrin and red corpuscles in the alveoli have disappeared, and their place has been taken by pus cells. At the same time the inflammatory changes in the alveolar walls and in the bronchioles may be more evident, and may have extended to the larger air-tubes.

Signs and symptoms.—The onset of the disease is almost invariably sudden. Thus in my cases there was a definite commencement in sixty-one cases; in three the onset was indefinite, and in the remaining one the child had been ill for three weeks, and the commencement of the pneumonia could not be determined. The attack begins with a feeling of malaise and generally vomiting. There is a cough and shortness of breath, and in children old enough to express their symptoms headache is a frequent accompaniment. The temperature rapidly rises, and the child soon becomes flushed and obviously ill. Food is refused, and a sharp pain in the side is often complained of. Herpes is sometimes present on the lips. The skin is hot, the tongue coated, and the respirations are quick, short, and "grunting." The pulse-rate is increased, but not in proportion to that of the respirations. The temperature rises to 104° F., or even higher, and the patient frequently becomes restless, sleep being disturbed by delirium. The signs continue severe till the crisis, when they rapidly subside. The crisis occurs in most cases between the 5th and 9th days. In my series the majority of the cases ended between the 5th and 10th days, the dates of the crisis being:

3rd day .	1 case.	10th day .	5 cases.
4th „ .	2 cases.	11th „ .	3 „
5th „ .	6 „	12th „ .	3 „
6th „ .	6 „	13th „ .	2 „
7th „ .	5 „	16th „ .	1 case.
8th „ .	4 „	17th „ .	1 „
9th „ .	4 „	18th „ .	1 „

In ten of my cases the termination was by lysis; nine of these were of short duration, the temperature reaching normal between the 5th and 11th days. In the remaining one the temperature did not subside until the 18th day; this was a case in which each apex was attacked successively. A few cases terminate as early as the 3rd or 4th day: they are generally mild, though the clinical signs indicate that the disease has passed through the usual stages. Other cases, on the contrary, continue as long as 18 days before the crisis occurs, and are

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to be explained by the disease not being localised in one patch, but spreading to other parts of the lungs. In a typical crisis the temperature falls abruptly from 103° F. or 104° F. to normal, with perhaps a slight rise on the following evening, but in children we frequently come across one or more pseudo-crises before the temperature finally reaches the normal line, and in this way a short lysis is produced. In young children again the temperature is often very irregular and remittent throughout the course of the fever. It may be 103° F. or 104° F. in the evenings and drop to nearly normal in the mornings, thus causing a difficulty in diagnosis if the signs are not distinct at first. Such cases, however, frequently end by crisis.

The physical signs indicating pneumonia are due to a diminished air entry and to consolidation of the lung. The shortness of breath and rapid respirations have been already mentioned. In addition to these you will often find cyanosis and recession of the intercostal spaces on one or both sides and in the epigastric angle. The movement on the affected side is less than on the other, and the percussion note is impaired, and in the later stages dull. The breath sounds are feeble or cut off at first, while over the rest of the lung they tend to be exaggerated. In this way the initial stages are apt to be mistaken for a pleural effusion. Later, however, as the consolidation becomes more marked the breathing has a tubular character, and bronchophony is present. Fine crepitations are also audible over the affected area; these may disappear as the tubular breathing becomes more marked, but reappear again towards the end when resolution begins to take place, and gradually become more moist in character as the condition of the lung improves. The vocal fremitus is often difficult to estimate in young children unless the patient cries. In the greater number of cases the disease terminates either in rapid recovery or in death. In the former case resolution is complete in about a week, and the child is soon restored to health. With the exception of empyema complications are infrequent. Long-delayed resolution is uncommon, and beyond some pleural adhesions, the result of a concomitant pleurisy, the after-effects are, as a rule, but slight. Death may be due to exhaustion, or to cardiac failure as the result of the specific toxine. Special watch, therefore, should be kept on the heart and pulse, for even quite early in the disease signs of cardiac failure may occur.

Complications.—Pleurisy is a condition which frequently accompanies pneumonia in children, and of itself rarely gives rise to more than a very slight effusion. Should, however, an infection by the pneumococcus or septic organisms occur, an empyema, as you know, often results, and in association with an empyema on the left side pericarditis may exist. Endocarditis is a rare complication, but meningitis or infection of the other serous membranes, such as the peritoneum or the synovial membranes of the joints, may develop. Otitis media and acute nephritis may also occur. Occasionally tuberculosis may develop as the result of a prolonged pneumonia, but this complication is not so common as in the lobular form of the disease.

Diagnosis.—The diagnosis between lobar and broncho-pneumonia is often by no means easy. A confluent broncho-pneumonia or an associated condition of collapsed lung affords very similar signs to those found in the lobar form. Some writers state that the occurrence of a crisis should determine the distinction between the two, but, as I have already pointed out, in some cases of lobar pneumonia in which the disease is extending the crisis is delayed, while in others the occurrence of pseudo-crises gives rise to a short form of lysis. The chief points of distinction to be borne in mind are: (1) That lobar pneumonia is almost always primary, while broncho-pneumonia, as we shall see, is very frequently secondary; (2) that its onset is sudden and its symptoms well marked, whereas broncho-pneumonia begins insidiously as a rule; (3) that the temperature tends to be more uniformly high, and to end by crisis, whereas in the other the course of the fever is prolonged and irregular and rarely ends abruptly; (4) that lobar pneumonia usually affects a localised area of one lung, at any rate to begin with, leaving the rest of the lung free, while broncho-pneumonia is bilateral and more extensive. With these points to guide us, and remembering at the same time that broncho-pneumonia tends to occur at an earlier age than lobar, we shall be able, in the majority of cases, to arrive at a correct diagnosis between the two.

The disease for which pneumonia is most likely to be mistaken is cerebro-spinal meningitis, especially in those cases in which pneumonia is accom-

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panied by marked cerebral symptoms. The temperature in meningitis, however, is generally lower than in lobar pneumonia, the pulse and respirations are slower and more irregular, and localised paralyses are apt to occur, all of which help the differential diagnoses. In those instances in which meningitis supervenes in a pneumonia the condition is generally marked by a sudden onset of cerebral symptoms in the course of the other disease. When such symptoms are present from the commencement they are usually transitory and due to the toxæmia. In the event of the physical signs of a pneumonia not subsiding we are confronted with the possibility of the presence of an empyema, for the recognition of which we must rely upon a flat or dull percussion note, absent breath-sounds or distant tubular breathing over the lower part of the lung with more definite tubular breathing above the level of the fluid, and displacement of the heart. In cases where empyema is suspected exploratory puncture should be made with all due care. Lastly, there is the possibility of pneumonia being mistaken for some abdominal condition. Typhoid fever with a more or less sudden onset and the early stages of a pneumonia, in which the history of the commencement is uncertain, are apt to be mistaken. Time alone will show by the development of pneumonic signs or the continuance of the fever without evidence of consolidation of the lung together with the presence of Widal's reaction which of the two the patient is suffering from. In some cases the pain in the abdomen and the rigidity of the abdominal walls very closely simulate acute gastritis or appendicitis, and it is only by carefully examining the chest that the true nature of the case can be made out. The usual error is to mistake pneumonia for some other disease, but considering the frequency of pneumonia in children we should be careful in excluding its possibility in any case with a high temperature and indefinite constitutional signs. Short rapid respirations combined with fever renders the existence of pneumonia highly probable. In those cases in which there is a doubt a thorough examination of the chest high up in the axilla, under the clavicle, and internal to the vertebral border of the scapula should be made, as in those regions an obscure pneumonia is likely to be missed.

Prognosis.—It is a good rule never to give up hope in a case of lobar pneumonia occurring in a child. To all appearances the patient may seem as bad as he can be, but with careful treatment and nursing he may ultimately make a perfect recovery. Out of my 65 cases 11 died, giving a mortality of 16·9 per cent. Speaking generally this may be considered as rather high. Of Holt's own cases only 11 per cent. died, while in 1295 collected cases be found the mortality to be only 3 per cent. With the exception of one case all my deaths occurred under the age of four years: two in the first year, four in the second, three in the third, and one in the fourth. In two the whole of both lungs was affected, in one both apices, in one the whole of one lung, while in the remaining seven the consolidation was situated in one or other of the lower lobes. The only death above the age of four years was the case of a boy, æt. 10 years, in whom the apex of the left lower lobe was affected. He died on the second day soon after admission to hospital, and in him the disease appears to have been of a virulent nature, as he went to school apparently well on the previous morning and was taken suddenly ill while at his lessons. Six of my cases died between the fifth and ninth days, of which one had both lungs affected, and one succumbed after the crisis as the result of an exploratory puncture for a supposed empyema. One case of double pneumonia died on the eleventh day just after the crisis, and another basal case on the sixteenth day. Two cases survived without crisis for three weeks, one of which was a double apical pneumonia, and one in which the whole of the left lung was involved survived for as long as four and a half weeks. The fact that no death occurred among those cases in which one upper lobe was involved deserves special mention, and points to a favourable prognosis in cases of apical pneumonia in children. It is also noticeable that in no case in which death was due to the disease did the temperature at any time rise above 104° F., in spite of the fact that five cases showed maxima between 105° and 106° F. and sixteen between 104° and 105° F., thus showing that the height of the temperature has little bearing on the ultimate issue, though a continued temperature above 105° F. should be regarded with apprehension as indicating extensive involvement or the presence of an accompanying pleurisy. Likewise

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a continuance of the fever beyond the tenth day suggests a spread of the disease or the presence of some complication. In a severe attack the outbreak of a fresh focus of disease in another lobe is always unfavourable.

The extent of the leucocytosis, which normally occurs during the course of pneumonia, has been considered by some as an indication of the severity of the disease, a severe infection inducing a violent reaction. It must be remembered, however, that a well-marked leucocytosis is partly the outcome of a strong power of resistance in the individual, and that a low leucocytosis may mean an enfeebled defence. Thus the true prognostic value of a leucocytosis may be masked. Generally speaking, however, the leucocytosis is well marked and increases with the area of lung involved, being greatest just before the crisis occurs. On the other hand, it appears that the extent of the leucocytosis bears no relation to the height of the temperature.*

Late convulsions are said to be unfavourable as indicating either exhaustion, toxæmia, or the onset of meningitis.

Speaking broadly the prognosis depends chiefly on the extent of the disease, and after that on whether there are any complications present. It must be borne in mind that the crisis is often a critical period, as signs of cardiac failure may develop just as the patient appears to be getting over his pulmonary disease.

Treatment.—Lobar pneumonia is a disease which in many cases tends to recovery of itself, and no form of treatment appears capable of aborting or shortening its course. The patient should, of course, be kept in bed in a well-ventilated room, and be disturbed as little as possible. If the temperature rises unduly high, or the patient is restless or delirious, cold sponging should be resorted to, and the same means may be employed to induce sleep, which is essential. If these fail to procure rest bromides are often efficacious. The cough is often scarcely severe enough to require expectorant treatment, but if sleep is disturbed small doses of heroin, or even of Dover's powder, may be used to secure the necessary rest. Pain, if severe, may be relieved by a mustard leaf or some counter-irritation to the chest. Signs of cardiac failure should be met by the administration of brandy, but the spirit should be kept in reserve until the necessity for it arises, *e.g.* at the time of the crisis. When it is called for I am more in favour of small doses, frequently administered, than large ones at long intervals. Subcutaneous injections of strychnine, either alone or combined with atropine, may be employed with advantage to tide the patient over a critical period, and oxygen is useful where there is cyanosis and much respiratory distress. Bleeding is very rarely necessary in children. In adults it is often of great service, especially in plethoric individuals, but indications for its use in childhood are seldom seen. Anti-pneumococcic serum has been recently introduced, but no great success has attended its injection, and I cannot recommend its use at present with any confidence.

* Heiman, 'Arch. of Pediat.,' 1905.

Broncho-pneumonia.

Broncho-pneumonia in childhood is more particularly a disease of early infancy. During the first two years of life by far the greater number of primary pneumonias are broncho-pneumonia, and throughout childhood nearly all the secondary pneumonias are of this variety also. My series of broncho-pneumonia cases numbers 85, and of these 33 were primary and 52 secondary, or 38·8 and 61·2 per cent. respectively.

Sex.—In my whole series the sexes were affected equally, 43 being males and 42 females. The same equality is also to be found when the cases are divided into primary and secondary, for in the primary cases I had 16 males and 17 females, and in the secondary 27 males and 25 females. In the secondary cases the sexes are affected about equally as a rule, but in the primary there is usually a slight preponderance of males.

Age.—The disease, as I have stated, occurs for the most part at a very early age, and after the fourth year of life tends to be uncommon. The ages of my patients were as follows:

Year.	Cases.	Deaths.
1st	15	9 (60 per cent.)
2nd	30	20 (66·6 ,, ,,)
3rd	15	4 (26·6 ,, ,,)
4th	11	2 (18·1 ,, ,,)
5th	7	2 (28·5 ,, ,,)
6th	4	0
7th	1	0
8th	2	2 (100 ,, ,,)

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It is thus seen that 60 cases, or 70·6 per cent., occurred during the first three years of life. Holt's statistics give the incidence of the disease as even earlier, for in his cases 86 per cent. were found during the first and second years.

Ætiology.—In contra-distinction to lobar pneumonia no one form of micro-organism is constantly associated with the disease. According to Wollstein * the pneumococcus is present in 76 per cent. of the primary and in 63 per cent. of the secondary cases. In many of the primary cases it is found alone, but often there is a mixed infection with the streptococcus, staphylococcus, or some other organism. In nearly all the secondary cases the infection is a mixed one. The cases in which the streptococcus is found are said to be the most severe, while the pneumococcus tends to produce areas of consolidation resembling in some ways true lobar pneumonia.

The chief predisposing causes of the disease are to be found in exposure and in the debility of the subject from inadequate hygienic surroundings, or from the existence of some previous disease. Thus in my series there were:

	Cases.	Deaths.
Primary . . .	33	15 (45·4 per cent.)
Secondary to bronchitis	7	3 (42·8 „ „)
Complicating whooping-cough . . .	34	15 (44·1 „ „)
Complicating measles	10	5 (50 „ „)
„ diarrhœa and vomiting . .	1	1 (100 „ „)

The greatest number of cases arises during the winter and early spring months, so that the question of exposure to chills is by no means unimportant.

Pathology.—The lesions as a rule are to be found simultaneously in both lungs; on only rare occasions is one lung alone involved. In my series of 85—

Both lungs were involved in 78 cases.
The right chiefly in 13 cases.
„ left „ „ 12 „
„ right only „ 5 „
„ left „ „ 2 „

The parts chiefly affected are the lower lobes posteriorly. You will find it rare for the anterior part of the lung only to be involved, but when this is so the right apex is the most frequent site. Here, as in lobar pneumonia, you will see that the tendency is for the right lung to be affected more than the left.

On section of the lung you will generally find evidence of a greater or less degree of bronchitis in the presence of secretion in the bronchial tubes, together with congestion and œdema of the lung. The essential lesions of broncho-pneumonia are small reddish or grey areas of pneumonic consolidation surrounding the bronchioles. These may be quite discrete or sufficiently confluent to cause consolidation of the greater part of one lobe. In cases supervening on a pre-existing bronchitis the inflammatory process descends from the larger to the smaller tubes, and finally affects the lobules in which they terminate. At the same time areas of pneumonia are often to be found in the tissues immediately surrounding the tubes throughout their length. In primary broncho-pneumonias the consolidated areas are also found in the immediate proximity to the bronchial tubes, which at the same time generally suffer from a concurrent bronchitis. In some cases there are also present scattered small areas of consolidation, chiefly towards the surface of the lung, which have no relation to the bronchial tubes.

Microscopically in the early stages of the pneumonic areas there is congestion of the smaller vessels together with extravasations of blood into the alveoli and into the interstitial pulmonary tissue. The air-cells are filled with red blood-corpuscles, leucocytes, and desquamated epithelium, with perhaps a little fibrin, while the bronchioles show a catarrhal bronchitis. Later, when to the naked eye the lung has a mottled appearance and the consolidated areas take on a grey colour, the air-cells are packed with leucocytes and pus-cells, as in the grey hepatisation of lobar pneumonia, and the surrounding air-vesicles show signs of early infiltration. The bronchitis is more marked, and pus is found in the lumen of the tubes. In the neighbourhood, and especially in the anterior parts of the lung a compensatory emphysema is also present.

Signs and symptoms.—In the majority of cases the disease begins insidiously, and this is especially the case in the secondary form. Of my 85 cases 61 had a gradual onset, and in 11 the commencement was indefinite; included in these are all but four of the secondary cases. In 13, or only 15·3 per cent., the symptoms began abruptly: these

* Wollstein, 'Montreal Med. Journ.,' June, 1905.

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consisted of 9 primary and 4 secondary cases. The onset in these cases is not unlike that of lobar pneumonia; the temperature is high, though not so uniform as in the other; the respirations are rapid; cough and prostration are marked, and the onset may be ushered in by vomiting. In the more insidious form the symptoms come on gradually. At first it may be difficult to determine whether there is more than a capillary bronchitis, but later more definite signs may develop. At the commencement the child shows a certain degree of malaise; the cough is frequent, and the respiration and pulse-rates are increased; the temperature is not very high, perhaps not above normal, and in the lungs are heard fine râles and a few rhonchi, either scattered over the back or limited to one base. After a few days the patient has more respiratory distress, the breathing is short and "catchy," and the alæ nasi begin to move with respiration. The cough becomes more distressing, and cyanosis of the lips and face may be observed. On auscultation fine crepitant râles are audible, with here and there patches of harsh or tubular breathing and slight bronchophony, while over the rest of the lung there may be coarser râles of a general bronchitis. Vocal fremitus is not perceptibly altered, and the percussion note may be only slightly impaired. The temperature tends to be higher, but as a rule it is of an irregular or remittent type, though a uniformly high temperature may be occasionally met with. In those cases in which a coalescence of the pneumonic areas occurs the signs approach those of the lobar form, but the dulness is generally not so marked, the lesions in the lung being more superficial, and the limitation of the affected area is not so sharply defined. Friction sounds from an accompanying pleurisy may be also heard.

Speaking generally, the temperature is not so high in this form of pneumonia as in the lobar type. Thus in my series a maximum of—

	Cases.	Deaths.	
105°–105·5° F. occurred in	5	3	(60 per cent.)
104°–105° F. „	„ 14	7	(50 „ „)
102°–104° F. „	„ 34	20	(58·8 „ „)
100°–102° F. „	„ 26	7	(26·9 „ „)
Under 100° F. „	„ 6	2	(33·3 „ „)

In two cases the temperature was normal throughout, and in two others it never rose above 99·6° F. As a rule the low temperatures are found in very young and debilitated children rather than in the older and more robust. The duration of the fever varies considerably, lasting in most cases from one to three or four weeks, but protracted cases may continue for three months or more. In my cases the duration of the fever was as follows:

Weeks.	Cases.	Weeks.	Cases.
1	9	8–9	4
2–3	15	13	3
3–4	9	14	1
4–5	12	15	1
5–6	9	16	1
6–7	5		
7–8	3	Doubtful	13

The termination of the disease is gradual in nearly all cases; in other words, a crisis seldom occurs. Only six of my cases, or 7 per cent., ended in this way: of these four were only of a few days' duration, one ended in the third week, and one in the fifth. Such cases are said to be pneumococcal in origin. Cerebral symptoms at the outset are not met with so frequently as in lobar pneumonia, but convulsions are apt to occur in the later stages, especially in conjunction with whooping-cough. Occasionally the disease, like the lobar form, may present a marked similarity to meningitis. Vomiting and diarrhœa are frequent accompaniments of severe cough, and the stools consist of undigested food and mucus. When these symptoms are severe and the patient is debilitated, they may be of grave import.

Complications.—Pleurisy and emphysema as accompaniments of broncho-pneumonia have been already incidentally mentioned, and are by no means infrequent. Occasionally the pneumonic areas may break down and form abscesses in the lung, which may go on to gangrene. With the existence of previous copious signs in the lungs such a condition may be easily overlooked. In acute cases purulent meningitis may develop. Rarely pericarditis complicates pneumonia on the left side. Stomatitis is not uncommon in infants, but other lesions of the alimentary tract are rare.

Diagnosis.—The chief points of diagnosis between broncho- and lobar pneumonia I have already dwelt upon. The distinction between broncho-pneumonia and capillary bronchitis is merely one of degree. The latter in all cases accompanies the former, and it is only by a continued or rising temperature and the development

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of signs of consolidation that a definite diagnosis can be made. In young infants prostration, cyanosis and rapid respiration should put us on our guard, even in the absence of other inflammatory signs, for these may not be present until after the lapse of a few days. Congenital atelectasis of the lung need only be considered during the first three or four months of life, but it must be remembered that the two may be associated. Atelectasis is only likely to be present in infants who have been weakly from birth, and have always shown feeble respiratory powers. The temperature is lower, and the cyanosis more marked than the physical signs justify. In the case of children who have been healthy, and have breathed well during the first month or two, the condition may be excluded.

There is one other condition which must be mentioned in connection with the differential diagnosis of broncho-pneumonia, and that is the tuberculous form of the disease, often called tuberculous broncho-pneumonia. This is especially important, as it is very liable to follow whooping-cough or measles, complicated by severe bronchitis or pneumonia, or in the course of an ordinary primary broncho-pneumonia. It is not possible to rely upon any particular physical signs for a diagnosis of the tuberculous condition. Judgment must be made on general grounds such as the family and personal history, the condition and environment of the patient, and the general course of the disease. The onset is generally very insidious, a period of malaise, wasting and fever preceding the onset of the pulmonary signs for some days, or even weeks. When the physical signs develop they are identical with those of broncho-pneumonia, and it is only when they fail to clear up that the diagnosis of tubercle can be made with any certainty, and even then we must remember that broncho-pneumonia sometimes develops into a chronic form. In the early stages the tuberculous character of the disease is very liable to be overlooked. If the patient has any other tuberculous focus or a dissemination of the disease, the existence of tuberculosis in the lungs is rendered more than probable. In suspicious cases the tuberculin test is a valuable aid to diagnosis, and I need only remind you of the convenient form of ophthalmic reaction, which has been lately introduced by Calmette.

Prognosis.—Broncho-pneumonia should never be considered lightly. A good deal depends upon the age and condition of the patient, his surroundings and his power of resistance, and upon the extent of the disease as evidenced by the physical signs. If a child takes his food well and has no diarrhœa or vomiting the case should not be regarded as hopeless. I had a total of 39 deaths in my 85 cases, or a percentage of 45·9. Of the 33 primary cases 15 died, or 45·4 per cent., and of the 52 secondary cases 24 died, or 46·1 per cent. My death ratio, therefore, was, to all intents and purposes, equal in the two classes, the very slight difference being in favour of the primary cases. Other authors, however, have found that the secondary cases are more often fatal than has been my experience. The highest death-rate in my secondary cases (not counting the one case complicating diarrhœa and vomiting) was after measles, 50 per cent. of such cases dying. This was closely followed by the cases complicating whooping-cough (44·1 per cent.), and those secondary to bronchitis (42·8 per cent.). During the first two years of life the mortality is high—60 and 66·6 per cent. respectively in my series. After that it gradually diminishes till the fifth year, subsequent to which death is uncommon (*vide* table, p. 374). A sudden onset of the disease in primary cases appears to be of unfavourable import, for of 9 such cases 5, or 55·5 per cent., died, 4 within seven days of the onset and 1 on the twentieth day. Among the secondary cases a sudden onset occurred in only 4, and of these but 1 succumbed.

The temperature affords some indication of the virulence of the infection. Hectic temperatures with high maxima are especially unfavourable, being due probably to a streptococcal or to a mixed infection. More uniform temperatures are generally pneumococcal in origin, and have a somewhat better prognosis. The relation of the death to temperature in my cases will be found in the table of temperatures (p. 376). There is generally a well-marked leucocytosis in broncho-pneumonia, but in this form it is independent of the amount of lung involved. It bears also no relation to the height of the fever, and diminishes at the same time as the pulmonary signs.*

Treatment.—As in all pulmonary conditions fresh air is essential. The child, therefore, should

* Heiman, *loc. cit.*

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be put to bed in a large, well-ventilated room, but in a position secure from draughts. Mild cases do well without any medicinal treatment, but in more severe cases expectorants, such as ipecacuanha and ammonium carbonate, are indicated, but care must be exercised lest they upset the digestion. Belladonna also is often useful in combination. Where there is much bronchitis light poultices, or a cotton-wool jacket, applied so as not to impede the respiration, are advisable; they should not be employed, however, if the temperature is high. Counter-irritation by means of a mustard application to the chest wall is also useful. Cough may be relieved by creasote inhalations from a steam kettle at frequent intervals, with just a curtain hung over the head of the cot, or heroin in $\frac{1}{100}$ to $\frac{1}{80}$ gr. doses may be given even in infancy for this purpose and to induce sleep. Opium should be very seldom used in the case of young children. With signs of cardiac failure or collapse stimulants, in the form of alcohol by the mouth or strychnine injections under the skin, should be employed. If the temperature is high, sponging I consider to be the best antipyretic; it also tends to reduce any nervous symptoms that may be present and to promote sleep. Care must be taken to maintain a good general circulation, and in the case of sudden collapse a hot mustard bath may bring the child round. The food should be light and administered at regular intervals, any over-distension of the stomach being guarded against, as thereby the cardiac and respiratory actions are liable to be hampered, and for this reason, if for no other, the bowels should be made to act regularly.

March 23rd, 1908.

ON INVALIDISM IN WOMEN DUE TO CHRONIC APPENDICITIS.

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THE application to gynæcology of modern surgical methods has in the past few years brought about a great reduction in the amount of chronic invalidism among women. The chronic invalid with a definite pelvic tumour has almost disappeared. There are still, however, many women martyrs to dysmenorrhœa and chronic pelvic pain, in whom no striking objective signs can be detected, and for this class surgery has, hitherto, done comparatively little. The object of this article is to point out that in a certain proportion of cases of this kind the symptoms are due to chronic appendicitis, and that they can be cured by appendectomy, if not by appropriate medical treatment. The main conclusions expressed in this paper were formed provisionally seven years ago. They have been tested to my own satisfaction by subsequent clinical experience in gynæcological and in general surgical practice.

If search is made in the text-books for mention of chronic appendicitis only the most cursory references to it will be found. In point of fact the disease has largely remained, even up to the present time, outside the range of practical diagnosis. It has masqueraded under various names—as chronic ovaritis, as ovarian or other forms of dysmenorrhœa, as cervicitis, as endometritis, as movable kidney, and probably as pelvic neuralgia.

The reasons for this want of recognition are not far to seek. In this country the practice of surgery and the practice of gynæcology have been sharply divided by the "scientific frontier" of the pelvic brim. When, as in the case of chronic appendicitis, a lesion exists on one side of the boundary, while its symptoms appear mainly on the other, a correct diagnosis becomes improbable, for neither a general surgeon nor a gynæcologist has a complete bird's-eye view of the whole field of disturbance.

Moreover, the physical sign upon which the diagnosis of chronic appendicitis must, in my opinion, mainly rest in doubtful cases, has been entirely misinterpreted. This physical sign is an indolent but intermittent swelling of the right ovary up to double its normal size. It is certain that the detection of such an enlargement has usually sufficed in the past to label the case as gynæcological, and to exclude the appendix entirely from consideration.

THE SYMPTOMS OF CHRONIC APPENDICITIS.

The symptoms of chronic appendicitis may be divided into three classes: (A) General, (B) Digestive, (C) Generative.

(A) *General Symptoms.*

Abdominal pain is a constant symptom. It may be shifting, but is felt usually in the right iliac fossa as a constant dull ache, subject to exacerbations upon any digestive upset. There is tenderness on deep pressure in the right iliac fossa as compared with the left one. In cases verging on subacute

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appendicitis slight muscular rigidity may be present on the right side.

There may be some loss of weight from imperfect digestion. The temperature may be slightly raised in the evening. Sleep is frequently unrefreshing, and disturbed by unpleasant dreams. Some patients complain of a lack of energy and of initiative, or of nervous prostration.

(B) *Digestive Symptoms.*

The digestive symptoms present little that is characteristic of the disease. Constipation may be present or absent. If it is very marked colitis should be suspected, and a sigmoidoscopy may be advisable to settle the question, for in colitis mere removal of the appendix may lead to disappointment. Diarrhœa may be present at intervals. There is usually dyspepsia, with flatulency, affecting chiefly the ileo-cæcal region, loss or capriciousness of appetite, furred tongue, and nausea. There may be an unpleasant taste in the mouth, especially on waking, or an unpleasant odour in the nostrils.

(C) *Symptoms arising in the Generative Area.*

Appendical dysmenorrhœa.—The most characteristic symptoms of chronic appendicitis originate in the pelvis, as abnormalities in the process of menstruation. The chronic right iliac pain already referred to is much intensified just before the onset of menstruation. Some dysmenorrhœal pain on the left side may accompany it, but the dysmenorrhœa of chronic appendicitis, in its characteristic form is felt exclusively on the right side, and in front of the abdomen. Appendical dysmenorrhœa does not date from the commencement of menstruation, but is of the acquired or secondary type. The pain most frequently commences at the same time as the menstrual discharge, but may precede it. At first very severe, it generally diminishes towards the end of menstruation. The flow is not necessarily increased, and may be diminished in amount. It may, however, be profuse and prolonged. One of my patients was kept in bed nearly half her time by prolonged and profuse menstrual periods. Since the performance of appendectomy her periods last only the normal time, and though still somewhat painful they do not confine her to bed even for a day. An even more characteristic symptom of chronic appendicitis is what I have called *dyspeptic dysmenorrhœa.* In cases of dyspeptic dysmenorrhœa a painful menstrual period is brought on prematurely, or is caused to return within a short period of its cessation as the result of an indiscretion in diet. For example, one of my patients, after an indigestible supper of veal, woke up in the night with acute right iliac pain, quickly followed by the onset of menstruation—four days before its expected time.

Alternate dysmenorrhœa.—In one of my cases the periods were alternately painful and painless. It is tempting to infer that the painful and the painless period correspond to ovulation in the right and left ovary respectively, but the value and frequency of this symptom are at present unsettled.

The Physical Signs of Chronic Appendicitis.

The right ovarian region is usually more tender to deep pressure than the left ovarian region. Right-sided muscular rigidity is only rarely to be made out. The principal physical sign of chronic appendicitis is what I have called the swollen right ovary. Other signs less frequently met with are thickening of the right utero-sacral fold and depression of the right vaginal fornix.

The swollen right ovary of chronic appendicitis.—It is not difficult to understand how the presence of an inflammatory focus in the appendix leads to congestion and œdematous swelling of the right ovary. A close analogy may be found in the "swollen face" of an alveolar abscess. Round any focus of chronic inflammation congestion occurs, and the tissue spaces are distended by an excess of lymph. These changes are passive and do not imply an active inflammation of the swollen part. Owing to the very rich vascular and lymphatic supply of the ovary, and to its peculiar tendency to the formation of small cysts, the œdematous changes show themselves first, and often exclusively, in the ovary. I have, however, several times detected other indications of œdematous swelling of the tissues around the appendix, especially a thickening of the right utero-sacral ligament and a definite depression of the right lateral vaginal fornix. Excessive secretion of cervical mucus may be another symptom of this congestion around the inflamed appendix.

The swollen right ovary of chronic appendicitis is usually of about twice the normal size, and is free from adhesions. It is often situated lower down than is normal, and may be prolapsed in the right

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lateral or posterior fornix. It has generally a thickened capsule of an opaque white colour. It usually presents numerous small cysts, prominent upon the surface, and containing clear fluid, to which the enlargement is partly due. Sometimes a large, bulging, dark-red area is visible upon the surface, and upon section a large recent hæmorrhage is seen, perhaps half to three quarters of an inch in diameter, evidently resulting from excessive hæmorrhage into a recently matured Graafian follicle. Or the effused blood may already show a convoluted layer of lutein at its periphery, resembling a corpus luteum of pregnancy. The excessive hæmorrhage which accompanies the maturation and rupture of a follicle, and the numerous small cysts, show that congestion and lymph stasis are the causes of the ovarian enlargement. Microscopically the ovary presents neither round-celled infiltration nor any other sign of active inflammation. It is to be noted that the swollen ovary is not, as a rule, particularly tender.

The enlargement of the ovary is an intermittent phenomenon, which may be present at one time and absent on a second occasion a week or two later. It may therefore be necessary to seek for it on more than one occasion.

The detection of the swollen right ovary.—The estimation of the size of the ovaries may, in some cases, be difficult or impossible without the aid of an anæsthetic. Even in simpler cases with thin and relaxed abdominal walls, the examination presupposes a tactile appreciation of the pelvic structures, which can only be gained by constantly renewed experience. The patient must be in the dorsal position. In the Sims' posture right-sided structures tend to fall towards the left, and an enlarged right ovary, if mobile, may be erroneously referred to the left side—a mistake which I have known made by an experienced gynæcologist. Lastly, in all cases a rectal examination should be made. An ovary which cannot be felt by the vagina may be easily palpable *per rectum*.

The enlargement of the right ovary possesses no significance unless it is so marked as to be unmistakable.

The frequency of chronic changes in the appendix.—Chronic inflammatory changes are frequently found in the appendix in women dying of other diseases. The appendix may be obliterated either at its tip, or along its whole extent. The submucous tissue may be thickened and fibrous, and the lumen, in consequence, may be much narrowed—a stage on the way to its obliteration. The epithelial lining may be entirely or partially absent, while the lumen contains a catarrhal secretion in which bacteria flourish. The lumen may be filled with soft fæcal material, or may contain concretions. Local strictures are not infrequent. The appendix mesentery may show a localised contraction, the result of a past cellulitis, producing a permanent and fixed bend or kink in the appendix.

So frequent are these changes in their slighter forms that some authors have considered them to be normal. It is to be granted that the slighter lesions of chronic appendicitis may exist without causing symptoms, and without doing any obvious harm, so long as the appendix can empty itself into the cæcum. In the same way a suppurating follicle in the tonsil may cause no trouble until its outlet becomes blocked. The introduction of virulent bacteria into the lumen of the appendix of a dog is not, as a rule, followed by any symptoms. If, however, at the same time the lumen of the appendix is constricted near its outlet, so that normal drainage becomes impossible, an acute attack of appendicitis results. The cases of chronic appendicitis which give rise to serious symptoms are:

(*a*) Cases of centripetal obliterative appendicitis in sensitive subjects.

(*b*) Cases of irregular obliterative appendicitis, in which an unobliterated part of the lumen remains as a closed cavity containing purulent fluid.

(*c*) Cases of catarrhal appendicitis with partial obstruction of the lumen from stricture, kinking, or concretions.

Sometimes an appendix affected with endo-appendicitis may be recognised as unhealthy by the congested condition of its peritoneal vessels, by marked clubbing of its tip, or by a sharp kink at one point along its length. But it must be expressly stated that chronic appendicitis rarely leads to the production of adhesions around the appendix. Without microscopic examination it is impossible to say upon inspection that any appendix is healthy and normal, even though it is absolutely unadherent.

I have seen an appendix so softened and infiltrated by inflammatory products that it broke

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off when pulled up into the abdominal wound, yet it had remained entirely free from adhesions. And on three occasions I have found localised collections of pus within the lumen of entirely unadherent appendices.

THE DIAGNOSIS OF CHRONIC APPENDICITIS.

The diagnosis of chronic appendicitis presents various difficulties. It will be well to consider briefly each of the conditions which may resemble it, and for which it may be mistaken:

(1) *Simple chronic constipation.*
(2) *Mucous or membranous colitis.*
(3) "*Chronic ovaritis.*"
(4) *Ascending salpingo öophoritis.*
(5) *Cervical endometritis.*
(6) *Movable kidney.*
(7) *Renal lithiasis.*
(8) *Gall-stone disease.*
(9) *Gastric conditions.*

(1) *Simple chronic constipation.*—It must never be forgotten that the commonest cause of abdominal pain in women is chronic constipation, occurring as a purely functional disorder. While constipation exists it is impossible to diagnose any other abdominal condition with certainty. The bowels must be cleared by purgatives and enemas, diet and exercise. If the pain disappears the diagnosis is clear; if not, further search must be made for its cause, one fallacy having been excluded. The more extreme forms of chronic constipation, which have been dealt with in such a bold and original way by Mr. Arbuthnot Lane, need not be here considered.

(2) *Mucous or membranous colitis.*—Obstinate constipation with abdominal pain may be due to membranous colitis. In such cases examination of the fæces will show membranous casts and shreds, sometimes mixed with mucus. There may be tenderness along the whole length of the colon. Examination by the sigmoidoscope, an instrument whose value Mr. Lockhart Mummery has so convincingly shown, may be necessary to clear up the diagnosis.

If membranous colitis is present chronic appendicitis is not thereby put out of court, for these two conditions are often associated. But in the presence of colitis the "swollen right ovary" loses much of its diagnostic value. It may be easily understood that a congested and chronically inflamed cæcum, just as readily as a chronically inflamed appendix, may cause œdematous swelling of the adjacent right ovary, and for identical reasons. Chronic appendicitis can only be diagnosed with certainty after colitis has been excluded.

How, then, are those cases to be dealt with where it is suspected that both colitis and appendicitis are present? Immediate appendectomy is to be deprecated, for though it may sometimes cure it is more likely to fail. In such cases a course of medical treatment for the colitis should be advised. If this fails to cure the colitis the next measure to try is an appendicostomy. Irrigation of the colon through the appendicostomy opening, and the administration by the same means of mechanical lubricants such as olive oil or vaseline, is usually successful in restoring the colon to health, and no further treatment may be necessary. Sometimes, however, the appendix, though it is dilated and at the same time drained by the appendicostomy, may continue to give trouble. I have seen an acute appendicitis following appendicostomy immediately and also at an interval of several months. In such cases when the colon has been restored to health appendectomy should be undertaken.

Cases of mucous colitis accompanied by diarrhœa require treatment on the same lines, save that here the colon may require mild astringent irrigations. I have had success with solution of tannin, one or two grains to the ounce.

(3) "*Chronic ovaritis.*"—A large proportion of cases of chronic appendicitis pass under the name of "chronic ovaritis." Chronic ovaritis, unaccompanied by adhesions, is probably a fictitious disease. It has no pathology, and denotes simply a combination of pain in the ovarian region with swelling of the corresponding ovary. Owing to the frequency with which the Sims' position is adopted for pelvic examinations, left chronic ovaritis is diagnosed as frequently as right chronic ovaritis, for in this position the right ovary falls over to the left side. In my own experience unilateral swelling of the ovary is right-sided in a majority of cases. If the other pelvic organs are healthy, if there is no evidence of ascending infection of the genital tract, and if colitis can be excluded, the detection of a "swollen right ovary" should not lead to the diagnosis of an imaginary "chronic ovaritis," but

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to the conclusion that chronic appendicitis is present.

(4) *Ascending salpingo-oöphoritis.*—The detection of a swollen right ovary is of no value as pointing to chronic appendicitis if there is evidence of ascending inflammation of the genital tract of gonorrhœal or puerperal origin. Consequently this sign acquires increased significance if the patient is a virgin. In all cases of suspected chronic appendicitis an attempt must be made to exclude salpingo-oöphoritis. This can only be done with certainty when there is an absence of muco-purulent discharge, when the mobility of the uterus is unimpaired, and when there is no thickening of the broad ligaments. That is to say, the swollen right ovary points unequivocally to chronic appendicitis only when other signs of pelvic disease are absent. Even a simple retroflexion is sufficient to deprive the sign of most of its value.

(5) *Cervical endometritis.*—I include this among the conditions from which chronic appendicitis must be diagnosed, because, in one of my cases, unsuccessful local treatment consisting of douching, painting, scraping and tamponage was applied to the cervix uteri for a period of over five years by various medical men and specialists for supposed cervical catarrh. The symptoms pointed to chronic appendicitis, and upon removal I found the tip of the appendix contained pus, though there were no adhesions. The symptoms at once disappeared, and have not returned.

It must be remembered that the congestion associated with chronic appendicitis may produce an excess of cervical discharge. The discharge, however, is clear and healthy when it enters the vagina, and only later becomes white from suspended vaginal epithelium. In true cervical endometritis the cervical discharge is muco-purulent before it enters the vagina.

(6) *Movable kidney.*—No one who has had out-patient experience in a hospital for women can fail to notice the extraordinary frequency of this abnormality. In the majority of cases it causes no symptoms whatever. It should only be diagnosed as a cause of right-sided abdominal pain when all the other possibilities have been as far as possible excluded, and when the symptoms are really characteristic. It is differentiated from chronic appendicitis by failure to detect the swollen right ovary, and by the absence of appendical dysmenorrhœa. The diagnosis of—

(7) *Diseases of the stomach and gall-bladder* from chronic appendicitis presents no special points, and cannot here be discussed at length. The positive indications of chronic appendicitis should, of course, be sought for wherever the possibility suggests itself.

Appendicitis causing chronic salpingitis and pelvic adhesions.—Many cases of chronic ill-health from matting of the pelvic contents by adhesions are due primarily to appendicitis. The view that nearly all these cases are due to the ascent of gonorrhœal or puerperal inflammation must be abandoned. During the past year I have three times had to operate for acute intestinal obstruction produced by inflammatory paralysis of the pelvic ileum. In all three cases the peritonitis was limited to the pelvis. In two of these cases the condition was primarily due to acute appendicitis, in the third to gonorrhœal salpingitis. In the causation of less acute cases of pelvic peritonitis it is probable that a similar proportion holds good, although actual proof is impossible in the absence of operation. Such cases later on no doubt often drift into chronic invalidism. They suffer, however, at this stage, not so much from appendicitis as from its sequelæ, and are therefore excluded from the scope of this paper.

In conclusion let me again remind you that some cases presenting the signs and symptoms of chronic appendicitis can be cured by a course of intestinal antiseptics and laxatives. The disease is one which should be treated by a physician before operation is decided upon. If colitis complicates the case the benefits of appendectomy, though sometimes great, are always uncertain. But in cases where colitis can be excluded, and where the patient presents the characteristic clinical picture I have described, appendectomy should be urged as promising complete restoration to health at the cost of a very small risk. The only serious danger of the operation, if due care be taken, is that of pulmonary embolism, which is fortunately a very rare calamity. Aseptic thrombosis of the femoral or saphenous vein, either of the right or left side, a not infrequent complication of appendix operations, can be averted by giving mild continuous circulatory stimulants during the period of convalescence. Two ounces of brandy or eight grains of ammonium carbonate per day, given every four hours in divided doses, will secure the end desired. It is probable that the same measures will succeed in warding off pulmonary embolism, which must be ascribed, like venous thrombosis, to circulatory stagnation resulting from prolonged recumbency. Lastly, if Battle's incision through the rectus sheath be employed, there is no need to advise the use of a belt, and no fear of a subsequent ventral hernia.

March 23rd, 1908.

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LECTURES
ON
CHRONIC INTESTINAL OBSTRUCTION.

By H. L. BARNARD, M.S.,
Surgeon (with charge of out-patients) to London Hospital.

Lecture III.

Intestinal Obstruction due to Compression by Tumours, etc., external to the Intestine.

In the cases brought together under this heading some large mass external to the intestines is supposed to press upon it and occlude its lumen. A few such simple cases no doubt do occur, but when I came to read the histories of some thirty-three cases which have been treated at the London Hospital it was evident in nearly every case that the direct pressure of the tumour had been complicated by adhesions, infiltrations, and inflammatory exudations which had taken a very large share in the obstruction.

Pathology.—Three factors take part in obstruction from compression. The coil of gut must be compressed against some bony surface such as the pelvic bones, the spine, or the ribs. In the second place the intestine must be fixed either by its normal attachments or by adhesions so that it cannot slip away. Finally, the tumour must either increase in size or become displaced so as to press upon the intestine. The acute symptoms are often due to the onset of inflammation about the tumour.

The above conditions most frequently coincide in the case of the rectum in the female, and the vast majority of cases of obstruction due to compression occur there from tumours of the female genital organs. Leichtenstern collected 165 recorded cases, and they were distributed as follows: In the rectum 60 per cent.; in the lower ileum 10 per cent.; in the splenic flexure, descending colon, and sigmoid flexure 12 per cent.; in the duodenum 7 per cent.; in the ascending colon and hepatic flexures 6 per cent.; in the middle part of the ileum 4 per cent.; and in the transverse colon 1 per cent.

Compression is a fairly common cause of obstruction, especially in women. At the London Hospital in thirteen years 32 cases occurred out of 669 cases of all forms of obstruction, and of these 29 were in women and 3 in men. The tumours causing compression are very numerous, and are best arranged according to the part of the bowel on which they press; thus—

The rectum has been compressed by cancer of the uterus, uterine fibroids, and retroversion and flexion of that organ. Ovarian tumours, especially when they are malignant, often obstruct it. Ectopic pregnancies, hydatids, and abscesses due to salpingitis or appendicitis, when they occupy Douglas's pouch, often compress the rectum behind and the neck of the bladder in front. Vaginal pessaries and vesical calculi, and sacculi, and sarcoma, and carcinoma of the prostate gland have also obstructed the rectum.

Tumours and abscesses arising from the pelvic bones and the sacro-iliac joint have produced a similar result. Finally, the cæcum and sigmoid flexures when loaded with fæces have descended into the pelvic pouch and compressed the rectum.

The ascending and descending colons may be compressed by retro-peritoneal sarcomata and other tumours; by cystic kidneys, hydronephrosis, or renal sarcoma or cancer; by abscesses around the kidneys, cæcum, or in the psoas sheath; or by tumours of the liver, gall-bladder or spleen.

The duodenum has been compressed by pancreatic cancers and abscesses or by lymphatic glands around the pancreas or in the small omentum.

The following case illustrates compression of the duodenum by extravasation of blood into the cellular tissue around it: A small boy was knocked down by a horse who planted a hoof in his epigastrium, as was clearly shown by the shape of the bruise. His abdomen was soft and was not tender, but his stomach became greatly distended and he vomited repeatedly. I opened his abdomen and found that his duodenum was buried in a large retro-peritoneal blood-clot. The peritoneum was intact. His abdomen was closed and he was fed by the rectum for a few days until the gastric distension disappeared, and he was able to take food without vomiting. He entirely recovered.

The superior mesenteric vessels and nerves sometimes compress the transverse part of the duodenum against the spine.

The small intestine may be compressed by

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mesenteric cysts, tumours, glands, and hydatids, by omental cysts and tumours, by floating spleens and kidneys, or a coil of gut may even be caught between the liver and the ribs.

Patel has stated that the intestine above a compression is dilated, but not hypertrophied unless ulceration occurs. It is, however, not uncommon to find ulceration of the rectum when that organ is compressed, and this leads to diarrhœa and even incontinence, which may complicate the diagnosis of obstruction in a most perplexing manner. I once had under my care a patient with a large hydatid situated in his recto-vesical pouch, which pressed upon his rectum. A large fæcal mass accumulated above the point of compression, and around this a stercoral ulcer formed some three inches in diameter, in consequence of which the patient suffered from persistent incontinence of fæces.

The symptoms of compression of the intestine from without present a good deal of variety. Nearly all cases at first present symptoms of chronic obstruction. The constipation is only partial, and some flatus and fæces are passed. Purgatives act, but only with difficulty and pain. Distension and flatulence are present and vary from time to time, and perhaps there is some vomiting, especially after solid food.

Many cases are, however, only seen when acute symptoms have supervened, and there is then often complete constipation, great distension, and persistent vomiting. In any case the tumour can usually be felt, especially if it is in the pelvis, and it is not difficult to surmise that it is causing the trouble.

The onset of the acute symptoms is due to some sudden change in the position of the tumour or to its rapid growth. In other cases a foreign body becomes lodged at the point compressed or the intestine above passes into a condition of paralysis and distension, and is kinked or twisted, whilst in not a few the acute obstruction is precipitated by the onset of local peritonitis around the tumour. The symptoms are never as acute as in those forms of acute obstruction in which strangulation is present, for in compression the mesenteric vessels are not greatly interfered with.

The treatment differs according to the situation of the pressure and the nature of the swelling. In many cases the tumour should be removed or emptied as in the case of the pregnant uterus or an abscess or hydatid. Where this is not possible a fistula must be established in the intestine above the obstruction. In a few cases it may be possible to establish a lateral anastomosis so as to short-circuit the part of the gut which is compressed.

The following case which I saw in consultation with a colleague illustrates very well many of the points about intestinal obstruction due to compression. A woman, æt. 31 years, was eight months pregnant when she was suddenly seized with acute colicky pain in her abdomen and became greatly distended. Her bowels were not open for four days and she was greatly troubled with "wind which she could not pass downwards." She vomited every day, more especially when she attempted to take food.

On examining her abdomen the uterus was found to reach well above the umbilicus, but its upper part was covered by distended intestine, coils of which could be clearly seen contracting; some of these were more than four inches in diameter.

On making a vaginal and rectal examination a soft swelling could be felt in the pouch of Douglas behind the uterus, and we correctly surmised that this was an ovarian tumour which had become incarcerated. The uterus was emptied by means of a tent, Hegar's dilators and a Champetier's bag and forceps, and the woman was immediately relieved of a vast quantity of flatus and several solid motions.

She did perfectly well and the child lived. Subsequently she was again admitted to the hospital and an ovarian tumour with a long pedicle which was twisted was removed.

March 23rd, 1908.

We have received from Messrs. C. Melin & Co., 37, Crutched Friars, samples of "Iola," which is a food that can be described as a highly nutritious concentration of nitrogenous, sugary, and phosphatised elements; it is a tonic and energetic stimulant, and not a medicine. "Iola" is invigorating and productive of heat and energy, and is a delicious condiment, easy to take, and to be recommended especially for invalids and persons with a delicate appetite. As no preparation is required, "Iola" will be found most useful at any time when need arises for sustaining or restorative alimentation. Clinical experience fully bears out the value of this new food.

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THE CLINICAL JOURNAL,

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A CLINICAL LECTURE

ON

STRANGULATED HERNIA.

Delivered at St. Bartholomew's Hospital.

By ANTHONY A. BOWLBY, C.M.G., F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—I suppose there is no subject of more general interest than strangulated hernia, and I want to say a few words on that topic to-day, because there are always cases in the wards, and there have been several instances lately, which are of, perhaps, more than usual interest.

Let me say a few words first of all as to what is meant by strangulation and how it occurs. A strangulated hernia is one in which the contents of the sac are constricted or strangulated, and that is the essential thing. These herniæ are often, indeed usually, irreducible, but the essential thing about them is that the contents are constricted, and the constriction is sufficiently severe, not merely to prevent the passage of the contents of the bowel, but also to interfere with the passage of blood, so that, as in strangulation produced by placing a ligature round a finger, there is first of all interference with the passage of venous blood, and then gradually as the part swells the constriction necessarily becomes tighter and tighter, and the tighter the constriction the more pronounced is the difficulty of the flow of venous blood, until finally the flow of venous blood becomes almost, or indeed completely, arrested. And before then the difficulty of the passage of arterial blood is also felt. That being the condition, then, you have to next consider what happens further as the result of this constriction of a piece of bowel. Not only do these changes take place in the bowel in consequence of strangulation and difficulty of circulation, but the bowel soon becomes inflamed, and the peritoneal coat

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exudes fluid. And that fluid, of course, collects in the sac of the hernia. The result in a very short time, in a few hours even in acute strangulation, is that the sac of the hernia becomes distended with fluid. At first the fluid which is in the sac is merely exudation from the peritoneal surfaces, but when any part is inflamed it is also softened, and you know that, as a result of inflammation, muscular tissue, fibrous tissue and submucous tissue may become so soft that the transudation of fluid from the inside of the intestine becomes easy. The intestine contains bacteria, and the consequence of this softening is that these bacteria percolate through the softened and inflamed intestine, and in that way they reach the cavity of the sac and contaminate the fluid which is in the sac, so that the sac itself becomes the seat of septic changes, and the whole wall of the bowel also becomes involved in a septic inflammation, in which, no doubt, the *Bacillus coli* plays a leading part. Supposing nothing is done for the patient, what more happens in these cases? We are at present speaking only of the local changes. If nothing is done, and the patient continues to live, the bowel will become more and more congested, until ultimately the circulation in it will stop. The circulation may stop at the one place where the constriction has occurred, and there a small piece of the wall of the bowel may slough, or the nutrition of the whole coil of intestine may be so interfered with that the whole coil sloughs or becomes gangrenous. Under such circumstances as these the contents of the intestine make their way out. So not only would the fluid in the sac be contaminated by bacteria, but also by the presence of fæcal matter from the inside of the intestine. And this condition would speedily give rise to suppuration, and, in some cases, to sloughing also of the sac and of the tissues outside the sac. And if the patient still continues to live, the result will be an abscess, with gangrenous contents perhaps, making its way to the surface. In such a case as that it is possible that the contents of the strangulated piece of bowel might ultimately be discharged, but it is very seldom that a patient lives so long as to permit of this final result being reached, although, as I shall presently tell you, this actually occurred in one of our patients recently.

That being the condition of affairs in strangulated hernia we have next to consider what these patients die of. I have already said two or three times "if the patient continued to live," but many of them die. A large number of patients with strangulated hernia would die before there was any suppuration of the sac; most would have succumbed before there was any sloughing of the intestine, and an important question is, Why do they die? What do they die of? In many cases the patients with this condition are old people, and the reason that they die is because strangulated hernia is associated with great pain and with inability to take or to retain food because of the sickness. These patients have distension of the abdomen also because, no fæces or flatus being able to pass, the abdomen becomes distended, and as a consequence of this the diaphragm is pushed up, and, abdominal respiration being made more and more difficult, the lungs become congested, and, especially in old people, these conditions are associated with some distension of the right side of a weak heart. That is how a large number of these patients die, and it is because of these complications, which have been established as a result of a purely local condition, and which remain in spite of your doing an operation for the hernia, that death ensues. Although you may operate successfully in plenty of cases of strangulated hernia, a large proportion of people will die so long as strangulations are left for long unrelieved; and the patients who are most likely to die are the old and the feeble, and they die in the way I have told you. So you see that unless the operation is done soon the patient will die quite independently of the local conditions of the strangulated hernia. Even if you operate upon patients before there is any gangrene, before there is suppuration, or even before there is severe sepsis, you will still find that a considerable number of them die, because they have been left, perhaps, two or three days, or even more, before any relief has been afforded, for an old person is easily killed by abstinence from food and by continuous sickness and pain associated with distension of the abdomen. Most of these cases are not found post-mortem to have any local lesion sufficient to account for death, which has occurred from complications following the local lesion rather than from the local lesion itself.

The next thing we have to think of is, What are the symptoms? How shall you know that the

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hernia is strangulated? The symptoms of hernia have been divided, and well divided, into those which are general and those which are local.

Let us look first of all at the local symptoms. What is it that attracts a man's attention to his hernia which has recently become strangulated? The first thing which it is necessary to realise is that, as a rule, when the hernia becomes strangulated the contents of the sac come down in larger quantity than usual, so that then the hernia is suddenly noticed by the patient to have become bigger than it usually is. And it is this increased quantity of the contents of the sac and the greater tension of the ring through which they are thrust which attract the patient's attention. It is this increased tension which causes the strangulation, and all strangulated herniæ are ordinarily larger than the patient has been accustomed to. That is the first thing. The next point is in regard to pain. The patient who has a hernia which is in the habit of coming down does not ordinarily have any material pain when it descends. Of course the man who had not had a hernia before and suddenly has a protrusion thrust out with some force through one of the rings has very acute, severe pain; but a person with a hernia which has been in the habit of descending frequently does not necessarily have pain unless the hernia becomes strangulated. Where does he feel this pain? The pain in these cases may be most misleading, because in all cases of hernia, and, indeed, in every kind of strangulation or obstruction of the bowels, some of the pain is referred to the umbilicus. In many cases the patient has urgent pain at the hernial region, but you must remember there are cases where a patient has no pain at the hernial region to attract his attention. And, indeed, there are cases where a patient denies having any pain, even if you closely question him. He will perhaps say: "I have no pain except at the middle of the abdomen," or the "middle of the stomach." And when people are fat and the hernia is small, if there is no local pain the presence of the hernia may be absolutely unsuspected, both by the patient himself, and, if the doctor is not careful, by him also. So there are plenty of cases where, in very fat people, a hernia, a femoral hernia in particular, which is often quite a small affair and covered over by large folds of fat in a pendulous belly, may be quite unnoticed, and if it is not sought for and felt for by the surgeon, who suspects that it may be present, and if he does not refuse to take the word of the patient for it, it may be completely overlooked. I have known the condition overlooked in such circumstances myself on several occasions.

Next, what local changes are to be noticed by the surgeon himself? The patient notices that the hernia is bigger than it has been before; he is aware of pain and he asks someone to see him. Therefore, when you examine him and feel the swelling what shall you feel for? The first thing which strikes a surgeon in the ordinary case of strangulated hernia is that as soon as he puts his hand upon it he feels there is greatly increased tension and hardness. An ordinary hernia is more or less soft to the feel. It is more or less, according to whether it has omentum in it or not, easily reducible by slight pressure. But when a hernia is strangulated you at once feel that it is a tense resistant structure. Why is it hard? What makes it tense? The great thing which makes for tension and hardness is the presence of fluid in the sac, and in proportion to the rapidity of the exudation of fluid and the retention of that fluid in the sac, the hernia feels hard. In a certain number of cases—a few—the hernia does not feel hard, even although it is strangulated. So that the mere absence of hardness or of tension does not necessarily mean that the hernia is not strangulated. In a few cases, as I shall have to tell you presently, the strangulation is *inside the sac*, by a band of adhesion for instance, so that the communication between the cavity of the abdomen and the cavity of the sac is not shut off. In such cases where the hernia is strangulated by a band of adherent omentum inside the femoral or umbilical region, if you open the sac you can put your finger into the cavity of the abdomen, and of course if that is so fluid can enter that cavity, and the sac is thus emptied. If there were no fluid to distend the sac there would be no tension in it, and it would not be hard. So there are a few cases—exceptional cases—where the hernia is not tense. But still what I said at first is true: the average hernia, when the surgeon puts his hand upon it, feels tense and hard, and that is the first thing which he notices. Again, the average strangulated hernia has no impulse on coughing. The strangulation gripping the gut at the neck of the sac or at one of the abdominal

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rings is so tight and holds the neck of the sac so firmly that no impulse can be transmitted from the cavity of the abdomen to the cavity of the sac, and consequently as nothing is thrust down in the act of coughing there is no impulse. But if the hernia is strangulated by bands of adhesions merely, not only may it not be tense, but there may be an impulse on coughing. Still, that is an exception. But it does not do to lay too much stress on these symptoms alone. You must lay stress on the other conditions, the general conditions, and chiefly upon the sickness. It is many years ago since Sir James Paget, lecturing here, said: "If a hernia has recently become irreducible and vomiting continues, that may be considered a strangulated hernia." And that is true still. It is one of those statements of truth which will last for all time—it will always be true. But you can see that a difficulty will come in in the class of patient who has had a hernia for a long time, and which has been irreducible for a long time, and who then becomes sick. Then you must consider the character of the sickness and the urgency of the sickness. In cases of strangulated hernia, generally speaking, the patient at first vomits the contents of the stomach, and afterwards, after a day or so, he regurgitates the contents of the duodenum, and later on he may regurgitate the contents of the small intestine, and ultimately the vomiting may be what is termed feculent, he will eject by the mouth the contents of the ileum. And in all cases of intestinal obstruction it is very noticeable that there are large quantities of fluid secreted by the intestine, and that the patient is sick out of all proportion to anything that he takes in the way of food. It is true that if he takes food he may be much more sick than if he does not take food, and there are cases in which a patient is very little sick unless he takes food. But, as I say, in many cases the amount of sickness does not bear a proportion to the amount of food taken, because large quantities of fluid are poured out into the intestines above the strangulation, and this fluid comes up in vomiting. Therefore it is the nature of the sickness, the character of the vomit, and the continuance of the sickness which suggests that there is something more than merely what is commonly spoken of as a bilious attack. So there are local and general conditions to guide you as to the probability that the patient has strangulated hernia, and not one which is merely irreducible. Now a word as to the intensity or acuteness of the symptoms in proportion to the contents of the sac. In proportion as the contents of the sac which are strangulated are small intestine, the symptoms are much more severe. If a person has a strangulated piece of the small intestine he is much sooner sick, and more urgently sick, and more often sick than if he has a piece of colon in it. And to such an extent, indeed, may the symptoms not be very urgent when the colon is strangulated that in not a few cases the patient will go on for a considerable number of days without being much sick when only the large intestine is involved. Thus, a patient was admitted into Stanley Ward eighteen months ago who had a strangulated umbilical hernia. She was a large fat woman, and she came in with a hernia which had been strangulated about four or five days; she was not quite sure herself how long it had been strangulated. When she came in and her clothes were taken off it was found she had a large piece of gangrenous intestine hanging out through the skin on her abdominal wall. There was a large area of sloughing skin which measured 4 or 5 inches across, and was perfectly dead and gangrenous. And underneath this gangrenous skin was protruding gangrenous intestine. And yet she was not sick or materially ill. All we had to do to her was to cut away some of this gangrenous tissue. Her whole transverse colon had sloughed, and she recovered like a person might upon whom colotomy had been done.

I mention that case to you to illustrate the fact that when the large intestine is involved the symptoms are often much less severe, and consequently more difficult to appreciate and diagnose, than in cases where the small intestine is involved. Occasionally omentum is the only structure involved in the strangulation. But you may say of strangulated omentum that it never gives rise to the same persistent vomiting or distension of the abdomen as does strangulated gut, either large or small, and that when omentum is strangulated, generally speaking it does not slough. Occasionally a small portion of it sloughs and a slow chronic abscess may form, and I have seen a patient who had evidently had strangulated omentum for something like five weeks, and when I operated all that I could discover was a teaspoonful of pus and a sloughing piece of omentum, which had to be cut

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away with scissors. The symptoms of strangulated intestine are not like those of strangulated omentum. In the latter the trouble is local; practically there are none of those general symptoms to which I have just alluded as characteristic of strangulation of the intestine.

Now let me allude to one or two cases in order to illustrate treatment. It is now about a month since, whilst I was in the operating theatre one afternoon, that I was told a baby had been admitted into the hospital with a strangulated hernia. When I heard that the patient was a baby of only a few months old and that the strangulation had only taken place a comparatively few hours before, I said that the best thing to do was to let the child be put into a cot on its back, and have its legs, thighs, and pelvis raised, and that someone was to sit beside it to keep it in that position. I left it there for perhaps an hour or so. Afterwards, when I went over to the ward, I found a little boy with strangulated hernia, but very little pressure and manipulation sufficed to return the contents of the hernia into the cavity of the abdomen. You will find a certain number of people at the present time are inclined to say that this manipulation, or "taxis," had better not be employed at all for strangulated hernia. That is a point of view I do not agree with in the least. It is said that in a certain number of cases harm is done to the gut, and that a surgeon cannot be trusted to use a sufficient amount of gentleness in reducing a strangulation. I very much question whether a person who cannot be trusted to be sufficiently gentle to perform taxis for this purpose is the proper person to be trusted to do an operation. And of the two I would rather trust him to do taxis than to do an operation. As a matter of fact the troubles which result from taxis are comparatively rare. Taking all the cases of strangulated hernia which you see in hospital, it is very seldom you hear of much harm being done by taxis, and I suppose that is because the average surgeon knows enough to appreciate the danger of using too much force. If you take all the cases of strangulated hernia in small infants, it is the rarest thing to have to operate upon them. I have never myself operated for a strangulated hernia in a child under a year old. I do not think I have operated upon a child under three years of age, and I do not expect to. The fact is that in practically all these cases of strangulated hernia in little children the position that I mentioned to you is alone sufficient, without any taxis at all. And if that is not sufficient, and the strangulation has only been in existence a few hours, then very little pressure, or taxis, is all that is required to complete the reduction. And that was all that was needed in this case. Now turn to patients at the other end of life—old people. Feeble old people with large herniæ almost always can be relieved by taxis unless strangulation has been in existence for a long time. You may wonder why I say "a large hernia." You may think that a large hernia would be naturally much more difficult to reduce than a small one. It is not so. In a small hernia presumably the parts around have not been much stretched. If a patient has a hernia of the size of a walnut in the femoral region, or in the inguinal region, in such a case the femoral ring or the internal or external abdominal ring or the neck of the sac has not been much stretched. But in proportion as the hernia has been there a long time and it has frequently come down and been replaced, and in proportion as the hernia has become large, these rings or apertures are stretched, so that not only is there more room for the hernia to come down but there is also, and for the same reason, more room for it to go back. So in old feeble people, and especially when they have large herniæ, taxis is in a very large proportion of the cases successful, in, I should say, the majority of cases in which it is applied during the first twenty-four hours. I will just say one word about how to apply it. In the first place I want you to clearly appreciate what it is you are trying to do, because unless you have a notion of how you are going to try and what you are aiming at, you are not likely to succeed. The first thing to do is to grasp the whole hernia and merely compress it firmly and gently, and not at first try to reduce it at all—simply compress it. It may be that the hernia is small enough to compress in one hand, or it may be a large one requiring both. In either case, your disengaged hand or the hand of somebody else should be employed in drawing down the hernia so as to pull it away from the abdominal cavity, and thus to pull open, as it were, the neck of the sac. So that in holding the hernia at the neck of the sac you try to draw it away from the patient's abdomen, while with the other hand

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you firmly compress it. What you are trying to do by compressing the hernia is to empty some of its contents. If there is any aperture through which the contents of the bowel can pass back so as to be transferred to that portion of the bowel which remains inside the abdomen, it is evident that by so much the size of the patient's strangulated intestine will be reduced. And that is true, not merely of fluid, but of the gaseous contents. So that by exercising pressure gently and firmly for two or three minutes you will in many cases begin to feel that the hernia is becoming a little less tense under your grasp. And in a certain number of cases no doubt the strangulated gut is also made more accessible to your fingers, because by your pressure you may gradually displace some of the fluid from the sac, and after two or three minutes of this steady pressure you can then begin to take hold of the contents of the sac with your finger tips and try and return them into the abdomen. And so long as you use merely the grip of your finger tips you will not do the patient much harm. Of course you must not lean upon the hernia or press any weight upon it, although it is astonishing what violence patients will sometimes inflict upon themselves in efforts at reduction, for they use an amount of violence which would not be justifiable in any surgeon. In the case of old people with large herniæ which have not been strangulated more than twenty-four hours, practically all of them may be safely treated in this way by taxis. But are there any cases of a different kind in which you are unlikely to succeed? Well, the cases in which you are least likely to succeed and which it is not worth while spending time upon are those where young, vigorous, muscular men have strangulated "congenital" herniæ. For even in a few hours such a hernia as that may be absolutely irreducible. A man with strong muscles has much more spasm and contraction of the abdominal muscles than an old person or a child, and in many of these cases where there is a congenital sac there is also a very strong, tough, fibrous ring which will not yield in the least Further, in proportion as the patient is young and the hernia has been thrust down with violence, as it often is in young muscular people, the strangulation will be acute and the exudation of the fluid will be very rapid. It is in these cases in particular that large quantities of fluid collect in the sac, and it is this large quantity of fluid which offers a mechanical obstruction to taxis, and often makes it impossible to return the gut. You will often find in operating on these cases that after opening the sac and letting out the fluid the strangulated piece of intestine goes back easily, perhaps with no division of the stricture at all, but merely because you have got rid of the fluid. So it is the quantity of fluid which is exuded in these cases of strangulated congenital hernia in young people, especially in the male sex, which is one of the chief obstructions to taxis. Then there are plenty of cases where you ought not to attempt taxis at all. A few weeks ago I came down here one evening to see a patient who was admitted on about the third day of strangulation of an umbilical hernia. This is the woman now in Stanley Ward. She weighed 25 st., and under those circumstances the hernia would be sure to contain a lot of fat, and this fat is as difficult to apply taxis through as is fluid, so that when a person is very fat and has a lot of omentum in the sac, there may be a knuckle of intestine there which you cannot influence by outside pressure at all. But this patient had had strangulation for three days, and I do not think it can ever be wise, when strangulation has persisted for more than two days, to try taxis at all. And in proportion as the contents of the small intestine are vomited, and in proportion as the patient is ill and the abdomen distended, it is not wise to try to put back the piece of intestine which may be in a doubtful condition. So without hesitation I operated upon that woman. When I opened the sac I found a lot of adherent omentum, and I found when I had got through that, just in the same way as when one had got rid of the fluid in the strangulated congenital hernia, there was no difficulty in getting back the piece of intestine which was there strangulated without dividing any stricture at all. And if you can do this so much the better for the patient, because in many cases where you have to divide the stricture it means that the patient has a greater tendency to hernia after the operation than he had before. But in the considerable majority of cases you do have a stricture to divide; the strangulating agent requires to be cut. You must know, therefore, what you are aiming at and what you are intending to divide when you start the operation. In the case of umbilical hernia the thing which you intend to divide if it is tight is the

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umbilical ring, and that is outside the sac altogether. In a case of femoral hernia the tight structure which you expect to divide is Gimbernat's ligament. In strangulated inguinal hernia the structure which you expect to find is most resistant is the neck of the sac itself, not as in the case of femoral and umbilical hernia something which is outside the sac. And in cases of this kind where you have to divide the neck of the sac itself you must divide it from inside the sac. But if you have a definite intention when you are operating upon strangulated hernia, your incision must be made in proportion or in relation to what you intend to cut. If a patient has a strangulated hernia, how shall you make your incision? The tendency, and, indeed, the temptation is to make the incision directly over the swelling. In umbilical hernia the lump may be large and protuberant—as big as a half-quartern loaf. You may say, "I must cut down into this." Do not. The right place to make your incision is at the part where the abdominal wall is continuous with the neck of the sac and not over the prominent fundus. In strangulated inguinal hernia with the scrotum greatly distended an incision in the scrotum is of no use at all; it must be made high up, close over the external abdominal ring and the inguinal canal. Let your incision be always where the neck of the sac is situated, because it is at the neck that you will expect to find the seat of strangulation. In a few cases you will not find it at the neck. There are those exceptional cases which I mentioned to you in connection with diagnosis, where the seat of strangulation is a band of some kind or another inside the sac. And that, of course, may be, and quite often is, in the fundus of the sac, and not at its neck at all. Those are quite exceptional instances however. As a rule the stricture which you aim at dividing is at the neck of the sac and consequently not in the most prominent part of the hernia.

And now I want to say a very few words about the intestine, and what you propose to do with it. Your intention as regards division of the stricture we have already alluded to. Suppose you have divided it, suppose you have divided the tight umbilical ring or Gimbernat's ligament, or Hay's ligament, or the external abdominal ring, or the neck of the sac as the case may be, and you have now sufficiently released the strangulated intestine to enable it to be put back, the temptation is to put it back at once; but it is a temptation which you must always be proof against. Remember that at the place where it has been strangulated it may have been hurt, and therefore before you put it back you must see the extent of that hurt. It may be that there is only to be seen on drawing it down and exposing it a line of slight discoloration to mark the place where the intestine has evidently been injured. Nothing more than that. You can put it back in that case. In other cases at the place where that mark is you will see that there is a portion which looks quite different in colour, perhaps a greyish or dirty grey or a purplish patch. And you say that it looks as if it were a slough already. It may be a very little thing, not larger than a split-pea. And you will ask yourself what is to be done with it—Am I to resect the intestine for that? It looks as if it might give way as soon as the intestine begins again to pass its contents along. Well, it may be that it is not safe to put it back like that, that it would be safer to treat it as if it were already a slough. And if you think it is not safe to put it back as it is, the right thing is to cover it in by drawing the peritoneum from each side over the small space which you fear may give way with two or three Lembert's sutures. But, and fortunately more rarely, the intestine as a whole may be gangrenous. You find a dead piece of intestine, and you have to consider what you must do with it. Let me remind you of one thing, which applies to all operations for strangulated hernia, and particularly in old people in whom the hernia has remained strangled for some time. Always be as quick as you can over what you have to do. This is not the best time nor are they the best circumstances for an elaborate operation for radical cure. It may be a very advisable thing for the patient to have a radical cure done, but when the patient has had a hernia which has been strangulated for two or three days, and where the operation of radical cure will occupy half an hour or more, you must remember it is not a favourable time to do an operation in a person who is already very ill. And the same applies still more in those cases where the gut is gangrenous. As a rule these patients are very ill and they are not in a condition to stand a prolonged operation. Curiously, however, some of them are not in a bad state, and I remember very well a man coming into the hospital from the country, having walked from

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Cannon Street Station, which is, I suppose, about half a mile from here, with a gangrenous femoral hernia. In a case such as that, where the man's condition is good, you can deal at once with the gangrenous intestine, cutting it away and then joining up the severed ends of the bowel. But in many cases people are too ill for that, and you must remember always that a prolonged operation may be the finishing stroke, and the mere length of the operation may determine the death of the patient. So if the general condition of the patient is good, notwithstanding the gangrene of the gut, it may be quite the right thing to operate at the time and cut away the gangrenous intestine, afterwards suturing the divided ends. But just as in the case of obstruction of the rectum due to cancer it is almost always wise to relieve the obstruction first by merely doing a colotomy, and then another day set to work to excise the cancerous intestine if it be removable, so also in these cases, in the large majority it is wise only to open up and relieve the strangulated intestine, allowing for a day or two free passage through the wound of the contents of the distended bowel, and then, if the patient can pull through that and is better, after two or three days—and the sooner the better—you can set to work to do the rest of the operation, joining up the severed ends of intestine and in that way preventing the formation of an artificial anus. Fortunately these complications are comparatively rare, and I think if there is one thing which is satisfactory about the hernia cases which one admits into hospital now as compared with the past, it is that these patients are certainly sent into hospital at an earlier stage than they were formerly. It was also a more common thing formerly to have these patients treated at home by pills and by castor oil than it is now. And when such patients were sent into hospital their condition was often such as to preclude practically all hope of recovery. But if you look at the percentage mortality of strangulated hernia now compared to what it was twenty years ago you will find that there are far fewer people who die after operation than there used to be, not, I think, because an operation is very much more skilfully done now or because we know much more about the after-treatment of these patients, but because a large percentage of the cases come in at a very much earlier period of their strangulation than they used to do formerly.

March 30th, 1908.

THE "PANCREATIC" REACTION IN THE URINE AND THE DIAGNOSIS OF PANCREATIC DISEASES.

By P. J. CAMMIDGE, M.B.Lond., D.P.H.Camb., etc.

ALTHOUGH diseases of the pancreas have received much more attention in recent years than was formerly the case, there is still a disposition to consider them as comparatively rare, and to regard their clinical differentiation as a matter of little practical importance. So long as the gross anatomical abnormalities, such as cysts and calculi, were considered to be the chief lesions of the gland likely to be met with in practice this might be true, but now that observation of the living subject during operations on the upper abdomen, combined with more careful and minute investigation in the dead-house, has conclusively proved that inflammatory affections are by far the most common lesions, and that many cases hitherto regarded as examples of "catarrhal jaundice," "chronic intestinal dyspepsia," "duodenal catarrh," etc., are in reality suffering from chronic pancreatitis, it is becoming increasingly important that more attention should be paid to the symptomatology of pancreatic diseases. Their clinical differentiation is now not merely a matter of academic interest, for the correct treatment of pancreatic disorders can only be based upon an accurate knowledge of their nature and ætiology. A striking example of this is furnished by the fact, to which Mr. Mayo Robson first drew attention, that many cases of advanced chronic pancreatitis are allowed to die unrelieved, under the mistaken impression that they are suffering from malignant disease. In some instances the symptoms are so similar, and even at operation the appearance and feel of the gland so much alike, in the two conditions, that it is only by exercising the utmost care in diagnosis, and by employing the information now to be obtained by an analysis of the excreta, that mistakes can be guarded against.

Disturbances of digestion due to pancreatic mischief are very rarely recognised clinically, but they are far from uncommon and account for a considerable proportion of those cases of chronic dyspepsia which do not readily yield to ordinary treatment. Unless the pancreatic element in such

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cases is recognised they may go on for years, and, in some instances, ultimately develop diabetes.

"Catarrhal jaundice" has long been the ever-ready explanation of cases of jaundice in which there were not gall-stones or other form of gross obstruction to explain the icterus, but it is now apparent that in all, or nearly all, of these the jaundice is due to pressure of an enlarged and inflamed pancreas upon the common bile-duct as it courses through the head of the gland. Chronic pancreatitis is found to be associated with the presence of gall-stones in the common bile-duct in about 70 per cent. of cases, and unless this is borne in mind the results of treatment are often not as satisfactory as they otherwise might be, and preventable complications and sequelæ are likely to ensue.

Among other conditions in which involvement of the pancreas often plays a part may be mentioned injuries of the upper abdomen, back-pressure from disease of the heart or lungs, arterio-sclerosis, blood infections, such as syphilis and pyæmia, cirrhosis of the liver and chronic alcoholism, gastric or duodenal ulcers, enteritis, sprue and other intestinal disorders, while inflammation of the pancreas may complicate mumps, typhoid fever and influenza. I have also seen cases diagnosed as malaria and pernicious anæmia which proved to be really pancreatic, and cleared up under suitable treatment.

Owing to the situation of the pancreas behind the stomach and in front of the spinal column, the physical signs of disease of the gland are often difficult to make out, particularly in stout people and in the early stages of the disease. The digestive symptoms are at times indistinguishable from those due to other causes, and are in some instances so masked by associated conditions that by clinical examination alone it is impossible to prove that the pancreas is the chief cause of the trouble. The relation of the pancreas to internal carbohydrate metabolism is now well recognised, and modern investigation has shown that about 70 per cent. of cases of diabetes are due to disease of the pancreas. Glycosuria is, however, a comparatively rare symptom of pancreatic trouble, and it occurs at such a late stage that to wait for its appearance is to throw away the patient's chance of cure. Maltosuria, pentosuria, indicanuria and lipuria, all of which have been suggested as indications of pancreatic disease, are in my experience so rare and inconstant that no reliance can be placed upon them in practice. From a general consideration of the history and symptoms of the case there is, however, often a probability, or strong suspicion, of some more or less serious interference with the functions of the pancreas, but in the absence of any definite indication by which this may be verified, the diagnosis must remain in doubt. Such conformation can, I believe, be obtained by a careful examination of the urine and fæces by the methods I have described, and particularly by the so-called "pancreatic reaction" in the urine.

The original method of performing the "pancreatic reaction," published in 1904 ('Lancet,' March 19th, 1904), has now been replaced by the "improved or C-pancreatic reaction," which I described in a paper read before the Royal Medical and Chirurgical Society on March 13th, 1906 ('Med.-Chir. Trans.,' vol. lxxxix, and 'Brit. Med. Journ.,' May 19th, 1906). The improved method is much more definite in its results than that first described, and is independent of the experience of the observer for its interpretation. The presence or absence of pancreatitis is now indicated by the result of a single preparation, and does not, as formerly, depend upon the difference in the amounts of the precipitates obtained in two parallel experiments. As it is a somewhat complicated process, however, it still requires some experience and skill for reliable results to be obtained.

I have now examined over 1500 specimens of urine for evidence of pancreatic disease by these methods, and have been able to confirm the diagnosis suggested by the examinations in a large proportion of the cases, either at operation or post-mortem. A detailed summary of the results obtained in 750 cases, together with the clinical histories of a number of them, is given in a recent work on 'The Pancreas, its Surgery and Pathology,' by Mr. Mayo Robson and myself, published by Messrs. Saunders and Co.

In many cases of pancreatitis, where recovery has followed drainage of the gall-bladder, and through it the infected pancreatic ducts, the gradual disappearance of the pancreatic reaction in the urine as the symptoms were relieved has served to indicate the dependence of the urinary changes upon the pancreatitis. In some cases it has been possible

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to re-examine specimens of urine several months, and in one instance over two years, afterwards, and they were still found to give no reaction.

The presence of gall-stones in the common bile-duct is not necessarily associated with pancreatitis, for according to Helly the duct is only embraced by the pancreas in 62 per cent. of cases. It is consequently of interest to note, as bearing upon the diagnostic value of the pancreatic reaction, that a reaction has been obtained in 65 per cent. of the cases of common duct cholelithiasis I have examined.

I have been able to test the findings of the pancreatic reaction in twenty-six cases by histological examination of the pancreas. In nine, where no reaction had been obtained during life, the gland appeared to be normal in every respect. In thirteen there were evidences of chronic inflammation, and a positive reaction had been obtained with the urines of all of them. Four proved to be cases of cancer of the pancreas. In two of these a correct diagnosis had been made, while in the other two the inflammatory changes accompanying the spread of the growth had pointed to a pancreatic lesion, but without an examination of the fæces it had not been possible to correctly determine the cause of the urinary reaction.

From the experimental side the diagnostic value of the test has been confirmed by Felix Eichler ('Berl. klin. Wochenschr.,' 1907, Nr. 25). This observer, in a recently published paper, states that he obtained a positive "Cammidgeschen reaktion," with the urines of three dogs in which acute pancreatitis had been experimentally produced, while the urines of normal dogs, also of guinea-pigs and men, gave no reaction.

It has been contended that the pancreatic reaction is due to the formation of lead salt crystals from the lead used in the neutralisation of the hydrochloric acid employed in the test, but that this is not the case is shown by the fact, among others, that in the improved reaction every trace of lead is removed by sulphuretted hydrogen. Others have stated that all normal urines give a positive reaction, but that is not my experience, and Eichler confirms my observations. The only feasible explanation of the unfortunate experience of the authors of these statements is, I think, that they had failed to carry out the various steps of the test in a satisfactory manner, and had not paid sufficient attention to the many small details which in this, as in most chemical investigations, are necessary for success. I have not, after over six years' experience of these methods, the least hesitation in stating that when the reaction is carefully and properly carried out by a competent observer it is of the utmost value in the diagnosis of pancreatic diseases, a positive result by the improved method indicating degenerative changes of an inflammatory character.

It is always advisable to check the results of the urinary examination by an analysis of the fæces, and in cases of suspected malignant disease it is essential for a correct diagnosis that this should be done, for although the improved pancreatic test gives no crystalline deposit in about 75 per cent. of cases of cancer of the pancreas, in the remaining 25 per cent. a more or less marked reaction is obtained, owing apparently to the inflammatory changes in the gland that accompany the rapid spread of the growth and blocking of the pancreatic duct. Mere inspection, and even microscopical examination, of the stools are not enough, the actual percentage by weight of the total fat, saponified, and unsaponified fats, in a carefully dried specimen must be determined, and the residue, after extracting with ether, be examined for stercobilin, preferably by the methods I described in 1905 ('Brit. Med. Journ.,' October 28th, 1905). In all cases of advanced and well-marked pancreatic disease there is, even on an ordinary mixed diet, a considerable excess of unabsorbed fat, often as much as 75 per cent. of the dry weight, but more important still the unsaponified fats, instead of being approximately equal to the saponified fats as in normal fæces, are in considerable excess. In cases of simple jaundice not associated with pancreatic disease the reverse is found, the saponified fats being here in excess. The total fats in cancer of the pancreas may reach 95 or 98 per cent. of the dry weight. In malignant disease of the head of the gland—the most common type—the fæces are quite free, or at most show but traces, of stercobilin at the time when the patient usually comes under observation, whereas in obstruction of the biliary passages by gall-stones, on the other hand, some stercobilin can nearly always be found, although the fæces may appear quite white to the naked eye.

My work upon the pancreas was originally com-

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menced with a view to finding some method of diagnosing a group of cases which is clinically most puzzling, and particularly with the object of differentiating cancer from chronic inflammation. The "pancreatic reaction" in the urine now enables us to recognise active inflammatory changes in the gland with a very considerable degree of certainty, and by combining the results of the "pancreatic reaction" with the information to be obtained by a careful quantitative analysis of the fæces, the presence or absence of malignant disease can usually be determined, particularly when the clinical signs and symptoms are also taken into account. I do not, however, wish to be understood as claiming that these laboratory methods of diagnosis are infallible, for there is a small proportion of cases in which the results are too indefinite or conflicting to permit of a definite opinion, but in the large majority of instances the correct diagnosis can be made, and in many the exciting cause of the pancreatic disease be determined, if one has had some experience of such work.

March 30th, 1908.

We have received from Mr. H. K. Lewis, of 136, Gower Street, a copy of 'A System of Radiography with an Atlas of the Normal,' by W. Ironside Bruce, M.D., Physician to the X-ray and Electrical Departments, Charing Cross Hospital, Hon. Radiographer to the Hospital for Sick Children, Great Ormond Street. The correct interpretation of radiograms is a subject which must engage the serious attention of practitioners, and an excellent effort to supply a book that will furnish them with a safe guide in intricate cases has been made by Dr. Bruce in his system of radiography with an atlas of normal bones. As the author, in his preface, very truly remarks, radiograms are too often valueless for diagnostic purposes from the fact that sufficient attention has not been paid to the relation between the source of the X ray and the part which is to be examined. If the normal is to be used as a means of comparison with the abnormal, it is absolutely necessary that both the normal and the abnormal should be taken with the tube in exactly the same relation to the part examined. Radiograms of the bones of the body at different ages are reproduced in full size, affording thereby a certain knowledge of the normal radiographic appearance. The book is a valuable addition to our means of diagnosis.

LECTURES

ON

CHRONIC INTESTINAL OBSTRUCTION.

By H. L. BARNARD, M.S.,
Surgeon (with charge of Out-patients) to London Hospital.

Lecture IV.

Chronic Intussusception.

I have had four cases of chronic intussusception; all have been operated on and all recovered. Two of these were in babies under a year old, one in a man æt. 29 years, and one in a woman æt. 40 years.

To give you some idea of the length of time a chronic intussusception may exist more or less unsuspected, I may state that my four chronic cases and two further ones which had become acute when I saw them had lasted eighteen days, three weeks, four weeks, six weeks, eleven weeks, and the longest nine months.

The two cases which after being chronic for a month only applied for surgical treatment when they became acute and distended, both died, one in five hours from the onset of the acute attack and before he could be operated on. The other died five hours after a Paul's tube had been fixed in a distended coil of ileum. It is therefore of the greatest possible importance that chronic intussusceptions should be recognised before they become acute, and I will narrate the two following cases in illustration of this point:

Case 1.—This man, G. H—, was 29 years of age. He was admitted into the London Hospital under a colleague, and was suffering from obscure signs of intestinal obstruction and an old hernia which was easily reduced when he was put to bed. This hernia unfortunately confused the diagnosis, as it was regarded as the cause of his symptoms. He had suffered for three weeks from attacks of griping pain and vomiting accompanied by the passage of repeated motions, but no note was made as to whether these contained mucus or streaks of blood. The abdomen was not distended, and as the

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hernia had been reduced he was watched for three days, during which the attack appeared to subside.

On the third day the colic and vomiting returned with greatly increased violence and the patient rapidly became distended. I was asked to see him and operate if necessary.

I found the patient had passed a motion both the day before and on the day I saw him. His abdomen was distended and contracting coils of gut were seen. He was vomiting continuously and his vomit was bilious. His condition was fair. I diagnosed a chronic obstruction becoming acute and operated at once.

The incision was the usual median one below the umbilicus. Distended and hypertrophied coils of small gut escaped at once from the wound. The sigmoid was found empty and the hernial rings were clear. A slack band of omentum was found adherent in the right lumbar region and this was divided, although it was not apparently obstructing the gut. No trace of the cæcum or of any collapsed coil of ileum could be found. I attempted to trace the gut down to the obstruction, but when I was nearing the lower end it passed high up into the upper part of the abdomen, and whilst I was endeavouring to draw it down the peritoneal coat ruptured badly, and as the patient's condition was very bad I inserted a Paul's glass tube at the point where the peritoneum had split and fixed it in the upper angle of my wound.

The patient died in five hours, and at the autopsy we found that the ascending colon had become invaginated into the transverse colon at the hepatic flexure, and had drawn after it cæcum, appendix, ileo-cæcal valve, and, finally, a piece of the ileum. The involvement of the latter was doubtless the cause of the acute termination of this chronic case.

The specimen is now in the London Hospital Museum.

Acute as the termination of this case was it was surpassed in this respect by the following:

Case 2.—T. T—, a man, æt. 37 years, complained of pain in his chest, in October, 1900, five months before I saw him. He was admitted to King's College Hospital, and his right pleura was three times aspirated and blood-stained fluid drawn off in quantity. Six weeks before he came to the Royal Chest Hospital he had suffered from obscure abdominal pains and vomiting, and had remained for some time in St. Bartholomew's Hospital. He was admitted to the Royal Chest Hospital for his lung condition, which was diagnosed correctly as new growth of the right lung. His bowels were opened as usual on the tenth day after admission, but he had slight abdominal discomfort. At 5 a.m. on the morning of the following day he was seized with violent abdominal pain, and his vomiting, which was continuous, rapidly became of a coffee-ground colour, and in a few hours he was distended and in a condition of profound collapse with a rapid and feeble pulse and a sub-normal temperature. I was sent for at 9 a.m. and reached the hospital ten minutes after he died at 9.45. His acute attack lasted but five hours.

The post-mortem was a remarkable one. He had primary new growth in the root of the right lung, and from this secondary deposits had been formed in all his abdominal organs except his spleen.

One of these secondary deposits had formed a polypus the size of a large hazel-nut in the ileum three feet above the ileo-cæcal valve, and this had led to the intussusception. Curiously enough, eighteen inches below the polypus and only just below the intussusception was a Meckel's diverticulum the size of the intestine about two inches long and free at its extremity. This had caused no trouble whatever. An almost identical case exhibiting new growth in the lung and a secondary deposit in the ileum producing intussusception was recently under the care of my colleague Dr. F. J. Smith.

The very obvious conclusion to be drawn from these two unfortunate cases is that chronic intussusception must be diagnosed and operated on in its chronic stage, and must not be allowed to become acute. The four following cases in which this was done all recovered, and complete the argument.

Symptoms.—It is then most important to recognise these cases, but it must be freely admitted they are most obscure. Rafinesque collected fifty-five cases, and of these no less than twenty-seven were incorrectly diagnosed. One of my cases had been treated for a month in the out-patients as a chronic colitis, and it was not until the ileo-cæcal valve hung pendulous four inches from the anus that the case was recognised and the sausage-shaped tumour found in the abdomen. Another was

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admitted under a surgeon, diagnosed as tubercular peritonitis, and transferred to a physician, under whose care the case remained five weeks before I was asked to explore the tumour, which I thought most probably carcinoma of the colon. It was an intussusception due to invagination of the caput cæci. Chronic intussusception has been mistaken for chronic colitis, cancer of the colon, fæcal accumulation, tubercular peritonitis, dyspepsia, gastro-enteritis, floating spleen and kidney, polypus and prolapse of the rectum, and many other conditions.

The onset is acute in about 30 per cent. of the cases; the symptoms then become alleviated and the case runs a chronic course. In 70 per cent., however, the onset is obscure and slowly progressive, commencing with colic and irregularity of the bowels induced by the taking of food or purgatives.

The diagnosis of chronic intussusception, however, turns almost entirely on the history of *repeated attacks of colicky pain.* When the patient is seen between these attacks little is to be discovered by physical examination unless a typical sausage-shaped tumour is present, and this was so in only three of my six cases. These attacks consist of violent colic, lasting usually several hours and waxing and waning in exacerbations. From the invaginated gut a copious flow of mucus is squeezed, and this, with perhaps a little blood, is passed at short intervals from the anus mixed with such fæcal matter as may have passed through the intussusception during the paroxysm, or may have reached the colon below before the attack began. This passage of several small motions during the attack, which is translated by the patient as "diarrhœa," is very liable to put one entirely off the track of an intestinal obstruction, but repeated attacks of colic with the passage of slime and perhaps of blood in one or more attacks is the great feature of this disease. *Vomiting* is quite an irregular symptom, and if it occurs at all is only during the attacks. It is more common when the invagination is in the small intestine or at the ileo-cæcal valve. *Constipation* is usually present between the attacks and the patients require purgatives, but, as I have said, during the attack itself "diarrhœa" is present. *A tumour* is present in the abdomen in half the cases, and it becomes larger and harder during the attack and passes further along the colon. Thus in one of my cases the tumour used to pass from the right loin across the abdomen until it reached beyond the middle line, and in another case of intussusception of the transverse colon near the splenic flexure it was only felt during the attacks as a sausage-shaped tumour in the left loin, and disappeared beneath the costal margin as the colic passed off. Between the attacks, when the spasmodic contractions of the colon have ceased, the tumour becomes softer and the natural attachments of the gut draw out the invaginated part so that the tumour once more retreats along the colon. It will be readily understood how difficult it is between the attacks to be sufficiently certain of the presence of organic mischief to proceed to open the abdomen. It is then that we appreciate the value of the scales which in all these cases tell us that there is very definite *wasting*, usually of more than a stone. In most of the cases there are signs of chronic obstruction. *Contracting coils of gut* can be seen on exposing the abdomen and perhaps kneading it, and the patient complains of *persistent rumbling of wind*, which may be a valuable sign in leading us to the point of obstruction where it stops. The patient becomes anæmic and exhausted as a result of the incessant pain, diarrhœa, and vomiting, and because the taking of food brings on attacks he starves himself.

Chronic intussusception sometimes occurs in babies, in whom it is usually mistaken for colitis, which, indeed, exists, but the most obscure cases are in adults.

Case 3.—The first case I ever encountered was in a male child, L. B—, æt. 1 year. His troubles followed measles. One month before admission he was seen by a general practitioner, who treated him for bronchitis and enteritis. At this time he vomited persistently for several days. Eighteen days before he came to the London Hospital he passed slime and blood with his motions, which were four a day, and this had continued ever since.

He screamed when passing each motion, which seemed to indicate violent colicky pains.

He had had a recurrence of vomiting (twelve times) the day before he was brought to the Surgical Out-Patients of the London Hospital, and this was the cause of his mother seeking advice.

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He was there diagnosed as colitis as he had just passed a motion of fæcal matter and slime, and was transferred to the physician, who, having found a large sausage-shaped tumour on the left side of the abdomen descending into the pelvis and palpable by the rectum, admitted him under the emergency surgeon for the day.

The abdomen, which was wasted, was not distended or hard, and after admission the child passed another motion containing plenty of bile and fæcal matter. The general condition was good.

The operation for reduction, which took but fifteen minutes from incision to dressing, was easy owing to the fact that the colon, which was flaccid and voluminous, had not grasped the intussusception at all tightly. This condition of atony was found in all my four cases, and no doubt accounts for the fact that motions passed freely along the lumen of the entering coil and that there were only traces of blood in the motions, for the intussusception could not be said to be strangled. It is, indeed, a true deduction that in intussusceptions the more the blood and the less the slime the tighter the strangulation is and the more acute and serious the intussusception; whilst conversely, the more the slime and the less the blood, and especially if fæcal matter passes with it, the less the strangulation and the more chronic the intussusception and the better in every way the prognosis. The last part of the intussusception formed by the invaginated ileo-cæcal valve was very thickened and œdematous, and in reducing it the peritoneum split in three places, but I did not stop to sew it up as some recommend, for the time so wasted would have seriously risked the patient's life. I have left such splits on several occasions and have never seen ill result. Except for the bronchitis which persisted and caused us some anxiety the child made an uninterrupted recovery.

A still more advanced case of chronic intussusception due to atony in a child is the following:

Case 4.—A. L—, a male child, æt. 11 months, suffered from diarrhœa seven weeks before he was operated on. For one month he attended medical out-patients for the passage of slime and a little blood with his frequent motions and for wasting. One day his mother brought him up to the out-patients with his ileo-cæcal valve hanging four inches from his anus. Through this he passed a motion. The index finger passed into the anus alongside the pendulous piece of gut could be freely swept all round it, clearly showing that this was not a case of *prolapsus recti*. A sausage-shaped tumour could be felt on the left side of the wasted abdomen descending into the pelvis. The child vomited but once after admission and passed another motion in the ward whilst awaiting operation.

I opened the abdomen opposite the umbilicus and just to the left of it, for this central incision enables one to pursue the intussusception round the circle of the colon, and is particularly adapted to manipulating it past the splenic flexure, which is often firmly fixed high up in the left hypochondrium. The rest of the colon is relatively freely mobile.

The hand of a dresser below the towels reduced the protruding gut and pushed it up the rectum so that my first and second finger, pinching the rectum between them could be passed below the intussusceptum. As these fingers were drawn up the rectum and sigmoid they pushed the intussusceptum before them. The rest of the intussusception was rapidly reduced, for the colon was flaccid, atonic, dilated, and there were no adhesions. When the cæcum was reduced it was found to be particularly flabby and capacious, but it was not even congested. The time of operation from incision to dressings was twelve minutes. The child made a good recovery.

An interesting little point in physiology and pathology is revealed by this case. Where the ileo-cæcal valve comes to lie in the rectum it will discharge its fluid contents in frequent small doses into that viscus, from which they will be discharged at once as very frequent small stools. Furthermore this explains the wasted shrivelled aspect of such cases, for under normal conditions these fæces would be deprived of their water in their passage along the colon and would reach the rectum in a consolidated condition.

The case I am now about to relate illustrates very fully the difficulties of these obscure cases, and is a typical story of their symptoms.

Case 5.—A. L—, a married women, æt. 40 years, eleven days before she came to the

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London Hospital began to suffer from attacks of severe colicky pains in her lower abdomen, particularly after food. This was accompanied by intermittent vomiting and her appetite failed. Her bowels were, however, regular and even relaxed.

On May 22nd, 1903, nine days before admission, she was seized with sudden and severe colicky pains and rolled upon the floor vomiting. She was treated for this at home, and when the attack passed off she applied to the hospital and was admitted.

Very little was discovered on examining her abdomen beyond the fact that she was tender below and to the right of her umbilicus. No doubt the intussusception at this time had reached the hepatic flexure and lay concealed beneath the liver.

A fortnight after admission, her symptoms having continued as before, an oval tumour was found below the right liver margin. This lump was freely movable and resonant on percussion. A physician was asked to see the case and concurred in the diagnosis of tubercular peritonitis, in which the omentum, thickened and rolled up, had produced the tumour. The frequent loose stools continued as before.

She lay for six weeks in the medical wards. Her appetite was wretched and she steadily lost flesh. The attacks of colic continued. She was constipated between the attacks, but during them her bowels were relaxed and her motions frequent. The tumour became much more distinct. At times it passed from the right border of the liver well across the middle line. Between the attacks it retreated to the right hypochondrium. It remained resonant on percussion.

At the end of this period I was asked to see the case as to the advisability of exploring the tumour. In examining the case I first excluded disease of the liver, kidney, gall-bladder, and stomach. No ascites was present, so that tubercular peritonitis seemed unlikely. The presence of contracting coils of gut, seen through the emaciated abdominal wall, and the persistent rumbling of flatus on the right side of the abdomen up to the tumour, through which it was heard, with a stethoscope, to squirt, considered with the resonant character of such a definite tumour, pointed to some disease partially obstructing the colon. Intussusception did not enter my mind and I thought the mass carcinomatous, but Mr. Hugh Lett, who saw the case as Surgical Registrar, correctly diagnosed it, basing his opinion mainly on the way the tumour advanced and retreated along the transverse colon.

The abdomen was opened just to the right of the middle line. An intussusception involving half the transverse colon was at once found and readily reduced, except the last piece, which was formed of the caput cæci. This was greatly thickened and indurated, and as often as it was everted sprang back again as though drawn in from within. I opened the cæcum some little distance from the caput, and a finger introduced showed that no polyp or tumour or ulceration was present, and the thickening was due to chronic inflammation. This opening was closed in the usual way by a double row of continuous sutures. The problem then arose as to how I should prevent inversion of the caput cæci, which occurred as often as I released it, in just such a way as a deflated rubber ball becomes dimpled in. I ran a purse-string suture of No. 1 silk round the base of the caput, and whilst this was held everted by the assistant I drew this sufficiently tight to prevent inversion but not sufficiently so to interfere with the circulation. This served the purpose admirably.

This patient's ileo-cæcal junction had a very long and loose attachment to the iliac fossa. It is interesting to notice that this mobility is indispensable for the formation of intussusception at this point, whilst in adults it permits of twisting of the gut and makes this the second commonest situation for volvulus.

The fourth case of chronic intussusception had many points of interest, for it was due to a myxomatous tumour of the splenic flexure of the colon, and the patient turned out to be a partial hæmophiliac. He subsequently developed a left subphrenic abscess in the blood-clot from the resection, but made a good recovery.

W. L—, was a gardener, æt. 29 years, and was sent to me by Dr. James Brown, of Tring.

Nine months before I saw him he had sudden severe pain in the left side of the abdomen whilst bending down, and passed a motion containing blood and mucus, but he did not vomit. There was no blood in the urine or frequency of micturition. He was quite well for a month after this,

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but suffered from constipation, and then another but slighter attack occurred and he again passed mucus without blood. After this the paroxysmal attacks of colicky pains became more frequent and he passed mucus in motions two or three times during these attacks, which lasted about five hours. He also suffered from flatulence which passed across his upper abdomen to his left side. He had steadily lost weight to the extent of a stone, and, owing to the persistent abdominal unrest, his appetite had failed. Between the attacks he had progressive difficulty in getting his bowels to act regularly, and he stated his motions were of small calibre. He had vomited but once during the first attack. No tumour or peristalting coils of gut could be discovered, but on one occasion an oval tumour was felt in the left loin, projecting below the ribs.

Definite organic obstruction of the colon appeared to be present, and I recommended exploratory laparotomy.

On December 28th, 1903, I opened the abdomen just to the left of the middle line. The sigmoid flexure was found empty and contracted. The incision was then prolonged to above the umbilicus and the transverse colon drawn out. This was distended. The hand was then passed up it towards the splenic flexure and a short intussusception was encountered. This intussusception was reduced by merely pulling strongly on the colon to draw it into the wound. It was then found that the gut contained an oval elastic body the size of a small egg attached to its inferior border, and that the gut was dimpled in on the external surface. The meso-transverse colon was thickened and indurated, and as I feared this infiltration was malignant I applied the gut clamps two inches on either side of the mass and removed it freely with the corresponding mesentery. The gut was united by a double row of continuous sutures. The oozing from the suture line was freer than usual and the abdominal wall also showed a tendency to ooze freely, but I did not pay much attention to this at the time.

He nearly succumbed to hæmorrhage after the operation. This was steady in onset and was not well marked for eight hours. His pulse-rate had then risen to 160 and was at times imperceptible. He passed repeated tarry stools, was restless, blanched, and suffered from severe headache. He was placed deeply under morphia, and the foot of the bed was raised about eighteen inches. Calcium chloride was given in large doses. He continued to bleed from the bowel for three days and then his pulse reappeared and slowed and he convalesced. On dressing his wound considerable oozing was found to have taken place into the dressings. I then questioned him and found that he frequently suffered from epistaxis, and that when his teeth were drawn he always bled for twelve hours.

There seemed no doubt that he was an ill-marked case of hæmophilia, but no other member of his family had suffered from hæmorrhage.

For a week he did very well, and then his temperature became slightly elevated and irregular and his pulse-rate once more rose. A fulness appeared in the left hypochondriac region triangular in shape, and limited by the middle line, the costal margin, and a line from the latter to the umbilicus. The interior of this triangle fluctuated and was resonant when the patient lay on his back, but was dull to percussion when he was on his side; from this it was evident the abscess contained gas. The lower edge was formed by a band three quarters of an inch wide towards the abdomen and was hard and tender. I have often found this definite tender edge with a painless fluctuating centre in appendix abscesses which are well localised.

When it was certain that he had a left subphrenic abscess in some trepidation I proceeded to open it by a vertical incision through the left rectus muscle. A large quantity of gas and pus escaped mixed with old decomposing blood-clot, and this was followed by a very copious and steady flow of blood. This was effectually controlled by packing the cavity with gauze soaked in adrenalin chloride and tightly bandaging round the upper part of the abdomen. When the gauze was cautiously removed after a day or two only moderate bleeding occurred, and the abscess cavity rapidly closed up.

The tumour was an ovoid and encapsuled mass lying in the submucous tissue and covered by the mucous membrane except where this had become ulcerated at one pole. It was succulent on section, and under the microscope was found to be entirely composed of myxomatous connective tissue. It contained no epithelial elements.

March 30th, 1908.

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The Index and Title-page for Vol. XXXI will be published with the next issue of THE CLINICAL JOURNAL.

THE OPERATIVE TREATMENT OF CANCER OF THE LARGE BOWEL.

By W. ARBUTHNOT LANE, M.S.,
Surgeon to Guy's Hospital, and Hospital for Sick Children, Great Ormond Street.

BEFORE considering the operative treatment of cancer of the large bowel, I would like to call your attention to the varying clinical behaviour of cancer as it affects the several portions of the large bowel. I do not propose to try to draw deductions from the vast literature of the subject, nor to accumulate statistics from my own practice or from that of other surgeons, but will confine myself to giving the views I have formulated from my own personal experience, and attempt to arrive at some general principles which may help to guide us in the direction in which we may do most good to our patients.

Generally speaking, when cancer affects that portion of the rectum which is not surrounded by peritoneum, its excision is less satisfactory than is the rest of the bowel. It is more difficult to remove the disease in its entirety, and recurrence is more frequent. This is particularly the case as the lower aperture is approached or reached.

Removal of growth invading the portion of the colon above this level is, as far as operations for cancer are concerned, a very satisfactory procedure if it is effected sufficiently early in the course of the disease, in that excision of the affected bowel and lymphatic glands may be complete while recurrence may be greatly delayed or avoided altogether. In the cæcum and ascending colon cancer spreads rapidly and affects glands at an early date, and over a rapidly increasing area.

Our prognosis must very largely depend on the state of the growth when it comes under observation, and the age of the patient is a material

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factor. The rate of progress of the disease and the tendency to recurrence after removal vary very greatly, though not uniformly in the several decades of life.

I should say it is very unusual for acute obstruction to ensue from growth affecting primarily that portion of the rectum that is uncovered by peritoneum, and this is probably true to a lesser extent of cancer starting in the cæcum. Cancer of the cæcum would appear to be more difficult to diagnose than that of any other portion of the large bowel. This is not due to its being inconspicuous, since it soon forms an appreciable mass. Yet I have unfortunately known such conditions watched carefully by men of great repute and experience during a period of time sufficiently long to render any attempt at removal hopeless, without any suspicion of the presence of cancer of the cæcum having suggested itself to them, though apart from the correct diagnosis very many others, often of a highly improbable nature, had been made and discussed.

It would seem that while in practice cancer affecting this portion of the large bowel presents much more difficulty in diagnosis than cancer of other portions of the large intestine, writers on the subject apparently have no difficulty in describing symptoms characteristic of its presence.

This would appear to be due to the fact that in the older subject the growth rarely interferes with the passage of fæcal matter from the ileum into the cæcum, while in the young subject the diagnosis of appendical trouble obscures the condition long enough to permit of very extensive infection of the lymphatic glands. Some inflammation of the appendix is not uncommon, particularly in the younger subject, owing to the occlusion of its orifice by growth, and if an abscess results the difficulties of diagnosis of the presence of growth are increased very materially, while any hope of the complete removal of the disease by operation is too often at an end.

Apart from the presence of a tumour suspicion as to the existence of cancer of the large bowel arises almost entirely from the mechanical interference with the lumen of the bowel. In some cases this may be a gradual process, the patient suffering from the frequent passage of fluid motions, which may be accompanied by blood and mucus, and by pain in the vicinity of the growth and in the portion of the bowel proximal to the obstruction, while in other cases, especially if the growth be at a sufficient distance from the anus to permit of the formation of solid fæces beyond the growth, acute obstruction is occasionally the first symptom of the disease. Yet such patients, when subsequently interrogated, will usually tell you that they have for some time noticed increasing fulness of the abdomen, griping pains, loss of weight, and show definite evidences of auto-intoxication. It is of vital importance that any evidence or suspicion of the presence of cancer of the large bowel should be gone into very thoroughly, so that delay in taking action and consequent useless regrets may be avoided.

The presence of a lump into which the bowel can be traced by careful manipulation is, of course, of vital importance, and knowing the value of time an exploratory operation should be undertaken at once in doubtful cases. The sigmoidoscope and the employment of X rays and bismuth, at one time administered by the mouth, and at another injected into the rectum, in a case where the obstruction to the passage of the bowel is insufficient to give decided clinical symptoms, are both useful, and may be employed with great advantage. Unfortunately it happens too often that when these cases come under the care of the surgeon the symptoms are such as to throw little or no doubt on the nature of the disease. This is, however, a question of education, and when the medical attendant has made himself more familiar with the condition, operation for cancer will be performed earlier and with happier results.

The operative treatment of cancer of the bowel, where it is covered by peritoneum, must depend enormously on the condition of the patient at the time.

If the symptoms are those of acute intestinal obstruction and the condition of the patient as regards the distension of the abdomen, toxicity, etc., is bad, the best course the surgeon can pursue is to tap the distended large bowel, or in the case of cancer of the cæcum the small intestine, at a convenient spot behind the obstruction. In certain circumstances, as in cancer of the sigmoid or transverse colon, it is sometimes possible to remove the growth which prolapses through the wound without unduly prolonging the operation. If, after recovery from this operation, the character of the

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growth suggests the possibility of its removal with any infected glands, this can be done and the remaining portions of large bowel brought into continuity by a lateral anastomosis. Later the passage of fæcal matter through the fistulous opening into the large bowel can be controlled by the use of a suitable shield, or the aperture may be closed by an operation.

Should removal of the growth be found to be impossible the ileum can be divided at its termination and connected by a lateral anastomosis with the large bowel beyond the growth, so enabling the patient to pass motions by the normal channel.

Should the condition of the patient suffering from acute obstruction permit at the time of the operation of the performance of either of these procedures, the annoyance and distress of a fæcal fistula and of a second operation are well avoided. Still, the surgeon must recognise that the risk entailed by doing this is proportionately greater.

Personally, my experience of the two methods of establishing continuity in the length of the large bowel is that the junction by lateral anastomosis is easier to effect and is more secure than the end-to-end variety. I do not mean that I would employ lateral anastomosis in every case to the exclusion of the end-to-end junction, but that generally speaking it entails less immediate and subsequent danger to the life of the patient.

If the patient be not suffering from symptoms of acute obstruction the intestines should be thoroughly evacuated aud their interior rendered as clean as possible by the use of internal antiseptics. One must be influenced very largely in the extent of the operative procedures, or in other words, in the immediate risk one is prepared to run by the age of the patient, by his circumstances, and by the wishes of himself and his relatives.

Should the sufferer be young, both he and his friends will doubtless be prepared to take very considerable risks, while on the other hand, if he is old and feeble, and if the duration of life is of great importance from a financial point of view, he and his friends may consider that a palliative procedure offers them all that can be expected, knowing that the palliative operation will, by removing irritation, render less rapid the growth of a cancerous mass, whose progress is often very slow towards the end of life. These points must all be carefully considered both before and during the operation, so as to ensure that the best possible is done in the interests of the particular patient.

Starting at the cæcum, if it be found possible and advisable to remove it, the cæcum and ascending colon and hepatic flexure should be freed from their attachments to the abdominal wall by the division of the peritoneum along their outer limit, and by separation of the bowel, by the fingers, from the loin. The peritoneum, which is reflected up internal to the bowel, must be carefully examined for glands, and the extent of their infection must be determined so that none shall be left which are possibly diseased.

Having got well beyond the limit of infection, the peritoneum is divided and the vessels in it are ligatured.

The transverse colon is surrounded by a ligature, the colon is cut through with a Paquelin's cautery proximally immediately in the vicinity of the ligature; the ligatured stump is inverted into the distal bowel and buried in it by purse-string and other sutures.

The ileum is ligatured at a safe distance from the cæcum, the absence of infected glands in its mesentery having been determined, the bowel is divided immediately beyond the ligature with the cautery, and the ligatured stump is buried in the proximal portion of the ileum.

The end of the ileum is approximated laterally to the side of the ligatured end of large bowel, and a large lateral anastomosis is established in the usual manner by a double row of sutures. Should complete removal of the cæcum with the infected tissues be considered impossible or inadvisable, the ileum should be ligatured proximally to the growth at two adjoining points; the intervening segment of bowel is divided with the cautery and the ligatured stumps are safely buried in the respective ends of the small intestine. The proximal portion is brought into apposition with the sigmoid or rectum and a lateral anastomosis is effected. By this last procedure the cæcal growth is freed from the irritation due to the passage of irritating fæcal material over its ulcerated surface, and any damming back of the contents of the small intestines which may have existed is relieved, so that the duration and comfort of the patient's life are both increased.

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In cancer affecting the several portions of the colon as far as the rectum the operative procedures are similar in principle to those employed in cancer of the cæcum, except that in the sigmoid, as in the portion of the rectum completely surrounded by peritoneum, an end-to-end anastomosis may sometimes be employed more advantageously than the lateral variety. This is particularly true of the rectal segment.

I would again call attention to the mode of fixation of the sigmoid which takes place when the obstruction is beyond this portion of the bowel, since a knowledge of its mechanics may be of vital importance to the surgeon. The causation and pathology are precisely the same as that described in chronic constipation.

Little tongues of peritoneal adhesions extend from the peritoneum lining the iliac fossa immediately external to the meso-sigmoid, and attach bit by bit the outer surface of the mesentery, shortening it up till the bowel no longer forms a loop but a more or less completely straight tube crossing the illiac fossa obliquely. Later the adhesions obtain a grip of the circumference of the bowel, commencing immediately outside its connection with the mesentery, and this fixation may go on till a considerable proportion of the circumference of the bowel is involved in the grip of these adhesions.

The bowel becomes shorter in proportion as its mesentery is shortened, so that when it is fixed firmly along the floor it no longer presents any evidence of a loop, but forms a straight tube. In extreme conditions of fixation its lumen is small and its muscle-wall thin. This condition has been noticed by many surgeons, and has been ascribed to numerous causes, none of which are, I believe, correct. Sometimes the mesentery has been regarded as congenitally absent. Usually the observer has been satisfied to note the fact without offering an explanation of its causation. As an illustration Schuchardt ('Centralbl. f. Chir.,' 1900, No. 28, Beilage) has emphasised the fact that in most cases of tuberculosis and of syphilis of the rectum or sigmoid, and in many cases of cancer of this region, the bowel becomes gradually shortened, and that this shortening may be very great, so much so as to obliterate the sigmoid loop.

Except in a stage of very advanced fixation it is possible to free the bowel and mesentery to a considerable extent by separating the points of attachments of the adhesions from the outer surface of the meso-sigmoid, during which process great care must be taken not to rupture any of the vessels supplying the bowel.

It is well worth the surgeon's while to make himself familiar with the arrangement and function of these adhesions, in order that by removing them he may be able to render the sigmoid and the upper portion of the rectum much more mobile, both for the establishment of continuity after resection of a portion of the bowel or for forming an artificial aperture in a convenient position.

The treatment of cancer involving the portion of the rectum which is not completely covered by peritoneum, and in which the symptoms are but rarely those of acute obstruction, must vary enormously with the circumstances I have already called attention to, namely, the age of the patient, the nature of the growth, the state of general health, the importance of duration of life to the individual, the distress experienced, and last, but not least, the intense objection which some patients have to the presence of an artificial anus. While one man is prepared to run any risk providing he is not left with an artificial anus, another man will readily acquiesce in its formation if he feels you have been able to eradicate the disease from his body as effectually as it is possible to do it. Personally my experience of an artificial anus made by leaving the divided end of the sigmoid opening at a point a little below and outside the umbilicus, is that the patient experiences very little discomfort indeed from its presence providing he wears a suitable belt and shield. The old-fashioned plug worn in an aperture in the iliac region was most uncomfortable and unmanageable, and was only preferable to the still older lumbar anus.

It seems to me that in a very large proportion of cases of cancer involving that portion of the rectum uncovered by peritoneum, as well as of the rectum in its immediate vicinity, the most important points from the patient's point of view are: First, that the disease shall be eradicated as effectually as possible, providing the age and medical condition of the sufferer render it advisable, and the disease is not too advanced, in which case a colotomy will give a maximum of benefit and relief at a minimum of risk.

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Secondly, that after the operation the patient shall suffer as little discomfort and pain as possible.

This last point is one that appears to me to receive much less attention than it deserves. We are dealing with a patient who may have suffered greatly physically, and who must of necessity since the knowledge of the existence of the disease have experienced and must continue to feel very great mental distress, since the public is fairly familiar with the pain associated with abdominal cancer in any form, and particularly with cancer of the lower part of the large bowel. The very term "cancer" is loathsome to them, and the probable uncontrollable escape of offensive discharge, or blood, or gas, or fluid motions, renders this condition particularly disgusting to people of refined temperaments.

Again there is that dread of infection that is creeping into our minds, and into those of the public, which is tending to render the lives of these sufferers even more distressing to them, and to those in their immediate vicinity.

In these circumstances it is of vital importance that the operation performed should at least not exaggerate the pain and distress already suffered, but should be of such a nature as to serve to remind the patient and those about him as little as possible of his trouble.

There is a certain proportion of cases in which a part of the lower rectum may be excised from the anus or by the vagina by Murphy's most clever operation, or by similar procedures. By such methods the control of fæces is left to the patient, and he leads his ordinary life. Such an operation is done at the risk of recurrence in the rectum and of progressive infection of the glands. Yet from the age of the patient, or from the very dense character and limitation of the growth and its chronicity, one is able to perform an effective operation and leave the patient in comfort. I have found that the use of X rays, properly applied, is of very great service in treating cancer of the rectum where the anal aperture is not involved. Mr. Shenton, our radiographer at Guy's Hospital, has been treating a number of such cases for me, both primary and recurrent, with remarkable success.

The method he adopts is to apply the X rays of high penetration (*i. e.* the rays produced by a tube of high vacuum) to the suspected region of recurrence or the site of residual growth, as the case may be. Daily applications of five to ten minutes are made for the first fortnight, followed by applications of the same duration on alternate days. Any sign of inflammatory reaction of X-ray origin is the signal for reducing the length of application. The period of time for continuing the treatment will vary in each case, but may be broadly stated as occupying a month. The tube used is sometimes specially made to suit a particular case, but the essential is adequate protection to surrounding parts. This is effected by suitable shields or by a tube made of glass X-ray opaque, except at the part in contact with the region treated. Overdosage of the rays from a low vacuum tube must be avoided, as causing inflammatory reaction, which will delay the course of treatment.

The success which has attended this treatment in his hands has encouraged me to have recourse on many occasions to operations which were not so complete as I would wish, while in early cases where removal of a small focus of disease involved an extensive operation because of its situation, I have been satisfied to leave the treatment to him alone and with the best results. In any case, however perfect the operation on the rectum, I like to have these cases treated by the X ray for intervals after the operation. This I do in all cases of cancer which have been operated on in order to reduce the risk of recurrence as much as possible.

When the disease is too extensive for these minor operative procedures, and when the patient is prepared to put up with the trifling inconvenience of a well-arranged abdominal aperture, the method I adopt is that of complete excision. I used to do it in one stage, but now prefer, if possible, to do it in two stages. By this the risk entailed is much reduced. The operation appears to me to effect the maximum of benefit to the patient with a minimum of risk and discomfort. The last feature, namely the absence of discomfort, is of paramount importance, inasmuch as the sufferer is already sufficiently depressed by the very serious nature of his malady. I only apply this procedure to cases in which it is not possible to resect a portion of the bowel to establish continuity. I make use of it in cases in which it is advisable to remove the growth and all infected tissues, and when the patient is prepared to accept the very trifling inconvenience

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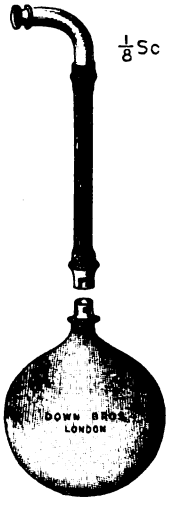
of a colotomy in the immediate vicinity of the middle line in the substance of the rectus. The younger the patient the more necessary is it that a very radical operation should be performed. The principle involved in this operation is the separation of the peritoneal space contained in the true pelvis with the diseased portion of the intestine from the general peritoneal cavity. This prevents any chance of a general infective peritonitis when the rectum is removed from below, and obviates the formation of adhesions involving the small intestines, since no raw surfaces are left in the pelvis to which the bowel can become attached.

The procedure is as follows: A vertical incision is made three quarters of an inch to the left of the middle line from the umbilicus downwards, for about four inches. The opening is formed in this situation, as it affords easy access to the pelvis and to the sigmoid flexure. In these cases the sigmoid flexure is almost always pinned down by adhesions. These are of the same nature, and perform the same function of obliterating the loop as in obstruction, due to constipation. A further reason is that in this situation a firm scar can be obtained in which the rectus muscle controls more or less completely the exit of material from the bowel. The patient being placed in the Trendelenberg position and the pelvic cavity being rendered fully accessible by the use of suitable retractors, the growth is clearly defined, together with any infected glands or mesentery. The sigmoid flexure is rendered mobile by the careful separation from it and from the meso-sigmoid of the acquired adhesions already referred to. A sufficient length of bowel having been drawn out of the abdomen, it is divided with the cautery at such a point as to leave two or three inches of the proximal bowel protruding from the abdominal incision after its edges have been approximated by sutures. The mesentery of the distal bowel is separated from the sacrum as far down as possible, and a ligature is tied firmly round the rectum at its lowest limit above the growth, any infected mesentery above the level of the ligature being thoroughly eradicated. The rectum is divided by the cautery immediately above the ligature. The ligatured stump is inverted into the rectum and securely buried by purse-string and other sutures. Then the peritoneum of the pelvis lining its wall and the bladder in the male, and the uterus and broad ligament in the female is picked up on a catgut suture so that that portion of the peritoneum of the pelvis which contains the rectal growth is completely separated by an artifical diaphragm from the general peritoneal cavity, and whatever disease still remains lies entirely below the protecting diaphragm which has thus been constructed. This is done easily in the female where the posterior surface of the uterus and broad ligament come readily into contact with the peritoneum covering the pelvic wall posteriorly. In the male subject, however, the formation of the diaphragm is accompanied with much more difficulty, and may make some demand on the skill of the operator. If, however, the patient is fairly upright in the Trendelenberg position, and the Doyen's

FIG. 1.



retractor has been removed, the posterior surface of the bladder can be sewn to the sides and posterior wall of the pelvis. The surgeon must remember that any defect in closure renders the patient liable to strangulation of a loop of bowel.

I find that the needle-holder which I use for cleft-palate operations is the most useful instrument that I know for the suturing required.

A curved tube fitted with a special rubber apparatus (Fig. 1), which I have employed for many years, is then tied into the portion of the sigmoid flexure left protruding from the abdominal incision, all fouling of the wound or discomfort to the patient during the whole period of convalescence being absolutely avoided by this means. The two or three inches of gut projecting from the wound is utilised to reattach the glass tube as each

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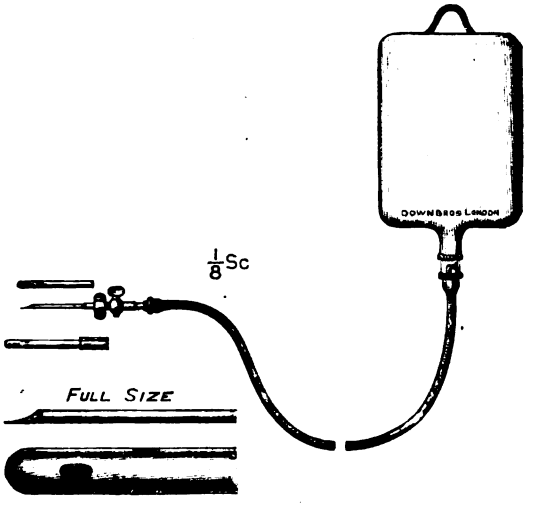
section of bowel sloughs off with the constriction due to the ligature till the bowel is divided flush with the skin. This leaves a perfect aperture which is readily controlled by the rectus muscle, and over which a cup and belt is worn without inconvenience.

After the operation the patient is put upon continual saline injections subcutaneously. Two pints are introduced into the axilla at once while the patient is still under the anæsthetic. During the three days following the operation about eight or ten pints are absorbed in this way by continued transfusion. To meet any risk of infection of the material introduced I devised an arrangement by means of which this can be effectually eliminated.

Fig. 2.



It has also the advantage of occupying a very small bulk in the bag of the surgeon. It is made for me by Messrs. Down Bros. It consists of a bag made of the best rubber capable of containing two pints of water (see Fig. 2). It has a metal orifice sufficiently large to admit of the ordinary salt "tabloids." To this is joined a rubber tube by means of a bayonet catch, to which a suitable needle can be attached. This needle is fitted with a shield, which serves the double purpose of keeping it covered after sterilisation and preventing any blunting of its point. A rectal nozzle is also supplied in the event of the surgeon wishing in certain cases to introduce the solution into the rectum. By using saline solution subcutaneously in this manner any sickness with the strain on the wound and the associated pain and distress is absolutely avoided, while the danger of shock to the patient from the operation is reduced to a minimum. I first employed this continual transfusion with the most marked success in cases in which I had removed the big bowel, with the result that the risks from the operation are very much reduced, while I believe the tendency to the formation of adhesions about the dilated small intestine, which is an occasional annoying sequence, and was, I believe, due to the fact that feeding was rendered impossible for a prolonged period because of the vomiting, is apparently obviated by it. The bag can be placed in any situation in or above the bed, and its contents can be kept warm by placing it on a hot-water bottle, etc. If the solution does not run freely pressure can be exerted on the bag readily or it may be suspended at a greater height. To employ it the bag is filled with water after the salt has been introduced into it. The shield, tubing, and needle are then connected, and the whole thing is put into any suitable vessel and boiled. The shield being taken off the needle, it is introduced beneath the cleansed skin, and is fixed in position with a suitable dressing so as to ensure asepsis at the seat of puncture.

After a fortnight has elapsed the remainder of the rectum is removed from below. This is done by a careful dissection of the rectum from the sacrum and from the bladder or vagina till the portion of the peritoneal cavity which has been shut off by the artificial diaphragm is encountered. From this peritoneal space into which the finger enters the remainder of the rectum is easily removed. The cavity left is drained in the usual manner and closes up rapidly.

One satisfactory result of this method of operating is that before the second step in the operation the patient gets about with comfort, and after the second step the sitting posture can be resumed after the lapse of a few days.

It may necessarily happen that owing to the presence of more than one growth in the large bowel, and especially if one be situated in the rectum, it may be necessary to remove more or less of the large bowel. These operations of extensive excision of the large bowel for growth are in my experience much more difficult to perform and much more serious to the patient than are the same operations for chronic intestinal stasis, yet

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this must not deter the surgeon from acting effectually in suitable circumstances. I once operated on a patient suffering from obstruction apparently produced by a growth in the sigmoid. She had had her breast removed for cancer many years previously, and there was no evidence of recurrence of the growth in the chest wall or glands of the axilla or neck or in the internal organs. On exploring the sigmoid the growth constricting the bowel was found to extend into it from its mesenteric attachment along a considerable length of the bowel. The meso-sigmoid was divided and the splenic flexure freed in order to enable me to form a lateral anastomosis between the transverse colon and rectum after removal of the intervening bowel, when I found another growth in the rectum and another in the ascending colon. All these were peculiar in that they invaded the bowel from its mesenteric attachment. I also felt one or two suspicious lumps deep in the liver. Having got so far there was nothing for it but to remove the entire large bowel up to the anus, through which the end of the ileum was brought and fixed there by sutures to the margin of the mucous membrane.

As far as the resection of the intestine was concerned the progress of the patient was satisfactory in that she could take nourishment and her bowels acted freely but loosely. She died within three weeks after the operation, her strength gradually failing, apparently from the spread of the disease in the liver.

If one had known that there was a secondary growth in the liver while excising the sigmoid one would not have proceeded to perform such a serious operation, but there was at first nothing to suggest it, and the obstruction had to be met with in some way. The report of the pathologist was that the growth in the mesenteric attachment was secondary, and involved in a varying degree almost the whole length of the large intestine. It extended into the wall of the bowel, reducing its lumen.

April 6th, 1908.

Infantile Scurvy.—Hamilton says that the anæmia, the restlessness or fretfulness when handled, the presence of blood in the urine, and unaccountable blood in the fæces, should lead one to look carefully at the food the child is taking, and should make one suspicious of early scurvy.—*Journ. A. M. A.*, vol. l, No. 12.

A CLINICAL LECTURE

ON

TWO CASES OF TRAUMATIC SPASTIC PARAPLEGIA.

By R. P. ROWLANDS, M.S.Lond., F.R.C.S.Eng.,
Assistant Surgeon, and Surgeon in Charge of the Orthopædic Department, Guy's Hospital.

GENTLEMEN,—I am bringing this fearful and crippling condition to your notice because it is very often believed to be beyond the hope of successful treatment.

It is caused by a destructive injury to the spinal cord usually in the lower dorsal region. A variable number of the nerve-fibres which descend in the pyramidal tracts from the motor areas of the brain are divided or severely compressed by a fracture dislocation of the spine, or by hæmorrhage into the spinal cord. When the injury is very severe and involves many fibres, the patient usually dies within a few months from bed-sores, pneumonia, cystitis, or ascending suppurative nephritis. For the few that escape these perils there is some hope of a slow regeneration of the compressed or divided nerve-fibres, but the recovery is rarely complete—more or less paralysis and spasm are usually left—so that the crippled patient joins the ranks of the unemployable. Regeneration and recovery may be made occasionally possible by a timely laminectomy designed for the removal of pressure upon the cord. But once spastic paraplegia with its characteristic deformities is well established neither *radical* operations, nor time and rest—those greatest allies of the healing arts—can remove deformities and disabilities which are dependent on unalterable structural changes in the nervous and muscular systems.

Even for these, however, *palliative* operations may do much—may, in fact, convert a cripple into a useful member of society—as you will see in the man that I am about to show you.

CASE 1.—George A—, æt. 26 years, railway labourer, was referred to me at the Orthopædic Department from the Electrical Department in June, 1906. He was then suffering from bilateral talipes equinus with slight cavus, and was only able to walk with the assistance of a stick.

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History.—On April 14th, 1903, he was knocked down by a train which struck his back. He was found in an unconscious condition, but when he reached Guy's he had recovered consciousness. There was much swelling and extravasation of blood over the spine, from the seventh dorsal to the third lumbar vertebra. No bony deformity could be discovered. The knee-jerks and plantar reflexes were absent. The legs were almost completely paralysed, but they gradually improved until the man was discharged on crutches at the end of two months. It was noticed that the spines of the lower two lumbar vertebræ and the first lumbar were prominent, and that the twelfth dorsal vertebra had been displaced slightly to the right.

The power and mobility of the feet had only partly recovered, the right less than the left. Soon after his discharge he found that he was unable to get his heels to the ground upon standing. For months the patient attended the Electrical Department, and the late Dr. Bryant recommended his re-admission into the surgical wards in February, 1904, but no operation was then thought to be advisable. He was referred back for electrical treatment. When admitted under my care on June 28th, 1906, both the feet were in a position of talipes equinus with cavus. When lying down the left heel was $2\frac{1}{2}$ in. above the plane of the ball of the great toe, and the right $3\frac{1}{2}$ in.

Upon pressing the fore part of the feet up it became clear that part of the deformity was due to spasm of the extensors of his ankles, for the deformity diminished; and when the man was standing his weight corrected the spasm so much that the right heel was only $1\frac{3}{4}$ in. from the ground, and the left heel just touched the ground. The anterior tibial muscles of both legs were somewhat wasted, the right more than the left. The wasting indicated a destructive lesion of either the anterior horn-cells or of a part of the cauda equina, in addition to the main injury. The knee-jerks were exaggerated and ankle clonus was present.

Operation.—An incision about 4 in. long was made along the hollow just in front of, and internal to, the right tendo Achillis. The tendon was exposed and slit longitudinally for 3 in. The inner half of it was then detached from the os calcis, and the outer half was divided obliquely 3 in. above its insertion. The two halves were then joined with catgut, the two ends being made to overlap one another with their raw surfaces in contact for about half an inch in order to obtain firm union. The right tibialis posticus was divided subcutaneously before the heel tendon was sutured, because the deformity was not completely corrected until this step was taken. The cavus was not enough to need division of the plantar fascia and plantar ligaments. The tendo Achillis and tibialis posticus of the left leg were then divided subcutaneously, and the feet were fixed at right angles.

The patient was discharged after three weeks. Since the operation he has always worn posterior splints with foot-pieces at night to prevent recurrence of the deformity.

For the left foot during the day he has also used an internal side-iron with a toe-lifting spring. He has been able to do his work upon the South-Eastern Railway since two months after the operation, earning the same wage as before the accident.

You will notice that his gait is almost perfect now without the instrument, which he will be able to discard after a few months without risk of recurrence. The power of the flexors of the left ankle has greatly increased since the operation, but much less improvement has occurred in the same muscles of the right leg.

Case 2.—Walter J—, æt. 25 years, was referred to me at Orthopædic Out-patients' by Dr. Fawcett, with the view of devising some apparatus which would enable the patient to walk. He was only able to go about on crutches owing to the extreme spasm of his legs, which were flexed and so strongly adducted that they crossed each other.

History.—On October 15th, 1906, he slipped off the steps of a moving electric car and was run over by a van; the horse also kicked him in the back.

He was taken into Guy's with a compound fracture of the right tibia and fibula. He was in the Hospital until the end of January, 1907, having been very ill with bed-sores and suppuration of the fractured leg. Years earlier the right knee had been excised for tuberculous disease, and the foot was rotated inwards as a result of the use of the vertical foot-piece instead of one properly rotated outwards to the normal position of the foot and leg at rest. While the patient was in the Hospital it was noticed that the injured leg was

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neither painful nor tender, and that bed-sores developed in spite of every care in nursing.

In 1907 he was admitted into the medical wards, and he then had all the usual signs of extreme spastic paraplegia. He was discharged *in statu quo*, and despairing of ever recovering his power of walking he has twice attempted suicide since then, although he is perfectly sane.

When I first saw him he had bilateral spasmodic talipes equinus, the left knee was semi-flexed and rigid, both hips were flexed and strongly adducted, so that it was almost impossible either to separate the thighs, or to extend them, or the left knee. The spasm was exaggerated by attempting to stand, by manual pressure upon the soles, and by any irritation of the legs.

Operation. — The two divisions of the right obturator nerve were exposed just below the obturator externus and excised. The tensor fascia femoris and the rectus femoris on the left side were divided subcutaneously.

On both sides the tendo Achillis and tibialis posticus were divided subcutaneously, and on the left side the semi-tendinosus was completely divided, while the biceps and the semi-membranosus were only partly severed, near their insertions, care being taken to avoid the external popliteal nerve. The result of these operations, which were all performed on the same day, will be mentioned later.

Diagnosis.

This is usually easy enough; some of the talipes equinus is due to spasm, and can be corrected by force; this and the absence of any considerable varus, valgus, or reaction of degeneration, distinguish the condition from all lesions of the anterior horn-cells in the cord, of the peripheral motor nerves, the muscles, or other soft parts of the legs. The facts that the deformity is bilateral and not associated with any paralysis of the upper extremities distinguish it at once from all forms of hemiplegia.

The real trouble in diagnosis is between this condition and congenital spastic paraplegia, so-called primary spastic paraplegia, and especially caries of the spine with pressure upon the cord. The history, if accurate, serves to exclude the former, for the symptoms of traumatic spastic paraplegia only appear after a severe spinal injury.

It is possible that primary spastic paraplegia may happen to appear soon after an accident, or that its symptoms may have been so slight as to be overlooked before the accident, yet a definite history of an injury to the spine before any symptoms are noticed is pretty conclusive evidence that the spastic paraplegia in these cases is really due to the injury. Moreover, it is not at all certain that there is such a condition as primary spastic paraplegia. It is more than probable that it is always secondary to some injury or disease of the upper neuron, although these may sometimes be overlooked or have disappeared before an autopsy is made.

Caries of the spine may follow an injury, and the cord may become compressed by exudation into the spinal canal, but this condition does not develop so soon after the accident, nor are its lesions so destructive and intractable as those of traumatic spastic paraplegia. Moreover, other signs of caries of the spine are usually only too evident, and the paraplegia is amenable to treatment by rest in the recumbent position. Such extreme spasm as crippled the second of my patients is not seen with caries of the spine.

Treatment.

Much can be done to prevent the development of deformities by carefully stretching the muscles, which are apt to retract as a result of chronic contraction: for instance, passive movements and a rectangular night splint may prevent talipes equinus in the early stages of the disease. When deformities have arisen, all that is necessary in mild cases is to divide the tendo Achillis subcutaneously, and to take precautions by means of apparatus to prevent the return of the deformity. It is gratifying to notice the great decrease of spasm that follows the removal of undue tension of the heel tendon.

When the talipes equinus is more extreme, as in the first patient under consideration, tenotomy may be followed by non-union and flail ankle, so that in these cases tendon lengthening is necessary, A real shortening of more than one and a half inches usually calls for tendon lengthening.

When the talipes equinus is corrected the anterior muscles are relaxed and allowed anew a range of movement which is essential for the recovery of their power. Massage, movements,

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both active and passive, and maintenance of the corrected position are material aids to the recovery. If the anterior muscles are paralysed and irrecoverable owing to destruction of their nerve centres in the cord, or of some of the cauda equina, the peroneus longus and a slip of the gastrocnemius may be grafted into the tendons of the peroneus tertius and tibialis anticus. In severer cases the spasm may affect the flexors of the knee, which may need division by the open method, which is much safer than tenotomy, especially as regards the external popliteal nerve, which lies in close relation to the biceps tendon upon its postero-internal aspect.

In others the tensor fascia femoris with the ileo-tibial band and the rectus femoris may require subcutaneous division for the correction of flexion of the hip-joint.

For adductor spasm the adductors have been divided only to rejoin and shorten again, as a rule, on account of the difficulty of maintaining the abducted position. Excision of these muscles has been more successful, but it is a severe operation which is not at all necessary, for resection of several inches of the obturator nerve is simpler, and not associated with either so much hæmorrhage or the same risk of suppuration in a wound placed so near the perineum.

In my patient I exposed and excised the two branches of the obturator nerve *below* the obturator externus, because I was anxious to preserve the external rotatory power of this muscle on account of the inversion of the foot, which had followed the use of the vertical foot-piece in the treatment of the fracture. It would have been easier, in this bulky man, to expose and resect a portion of the nerve-trunk in its intra-pelvic portion. A vertical incision through the lower part of the rectus muscle, with displacement inwards of the parietal peritoneum, allows a fairly easy and almost bloodless approach to the nerve as it lies on the outer wall of the true pelvis in front of its corresponding vessels.

The right nerve was chosen because the right leg was $3\frac{1}{2}$ in. shorter than the left, as a result of the compound fracture of the leg and the excision of the knee for tuberculosis some years earlier. The results of paralysing the large adductors of this patient on the right side only are that the thighs no longer cross each other, but can be flexed without touching, and that the right lower limb has become abducted enough to make it an inch longer for the practical purpose of walking, so that cork in the boot has been shortened to that extent. Further, the spasmodic flexion of the right thigh, which was never so marked as that of the left, has been almost abolished by putting the adductors out of action.

The man can still adduct his right thigh into contact with the left chiefly by means of the pectineus, and the hamstrings which have acquired new insertions about the excised knee. The result of the multiple tenotomies and division of muscles has been the correction of the equinus and of the flexion of the knees and hips. Even a more striking thing is the abolition of the spasm of the affected muscles, although their tendons have united. Care has been taken to prevent shortening of these tendons during the contractile stages of healing, so that they are no longer subject to abnormal tension, which is a potent cause of reflex spasm.

The net result of all the operations is that the patient is now walking a little with the aid of two sticks with the feet flat upon the ground, and without any instrumental support. He is no longer depressed, but hopeful of further improvement, for only three months have elapsed since the operations. It is of importance to remember that the wounds heal naturally in patients with spastic paraplegia if proper aseptic precautions be taken. The risk of sloughing and of the development of pressure-sores has been greatly exaggerated, but they clearly exist in early stages of the disease.

April 6th, 1908.

THE result of the opsonic treatment of gonorrhœal vulvo-vaginitis in a small hospital epidemic, involving twelve children of ages ranging from one and one-half to twelve years, is reported by W. J. Butler and J. P. Long. The gonococcus was readily grown on blood serum and blood agar. They found a considerable advantage in using young cultures from six to eight hours old, to avoid trouble from clumping, and consider this an important point in minimising the difficulties of opsonic work in general. This holds good in the preparation of the vaccine as well as that of the emulsion.—*Med. Record*, vol. lxxiii, No. 12.

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LECTURES
ON
CHRONIC INTESTINAL OBSTRUCTION.

By H. L. BARNARD, M.S.,
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Lecture V.

Simple Cicatricial Stricture of the Intestine.

This is an exceedingly rare cause of chronic intestinal obstruction. The surgical registers of the London Hospital contain 669 cases of all forms of intestinal obstruction in 13 years, and of these only 13 were due to simple cicatricial stricture.

Cicatricial strictures may arise from the contraction of scars left by ulcers, or they may follow limited gangrene or sloughing such as may result from trauma or strangulation. In some cases due to syphilis and in the hyperplastic form of tubercular stricture the fibrous tissue is deposited in the layers of the gut without any loss of substance.

Cicatricial strictures may be classified thus:

(*a*) Those due to some form of ulceration.

(*b*) Those which follow abdominal trauma.

(*c*) Those which result from strangulated hernia.

(*d*) Those which follow the spontaneous elimination of an intussusception.

The 13 cases at the London Hospital were distributed as follows: (*a*) *Ulcerative* 9—that is, tubercular 4, syphilitic 2, duodenal ulcer 1, undetermined 2; (*b*) traumatic 3; (*c*) strangulated hernia 1.

The Anatomy and Pathology of Cicatricial Strictures.

Of 13 cases of cicatricial stricture which occurred at the London Hospital 4 were in the sigmoid flexure and 9 in the small intestine.

It has been stated by Nothnagel and Sir Frederick Treves that this form of stricture is more common in the colon than in the small intestine, and the latter has stated that the ratio is as 6 to 1. These authorities, however, appear to include simple strictures of the rectum in their figures, where syphilitic dysenteric and traumatic strictures are relatively common, and they apparently regard a narrowing of the lumen of the gut as a stricture whether it produced symptoms of stenosis or not.

A cicatricial stricture may be quite short, as though a string had been tied around the intestine. In other cases where extensive ulceration has produced the stenosis the stricture is long and tubular. In syphilitic and tubercular cases a considerable tumour often exists through which the narrowed lumen of the intestine runs. The degree of contraction necessary to produce obstruction depends upon the nature of the intestinal contents. In the small intestine no obstruction is produced until the lumen is nearly obliterated unless a foreign body becomes impacted. In the lower part of the colon, however, where the contents are normally solid, a very moderate degree of stenosis is sufficient to lead to intestinal obstruction.

A cicatricial stricture is surrounded by adhesions and much organised lymph, which unite it to neighbouring structures and take a part in the contraction of the lumen.

A simple stricture is formed of pigmented cicatricial tissue in which patches of round-celled infiltration frequently occur. When a tubercular lesion has produced the contraction, giant cells and tubercle bacilli can usually be found in the section. When the stricture is syphilitic, the arteries show the changes characteristic of endo- and peri-arteritis. The intestine above a stricture is hypertrophied, dilated, lengthened, and spreads between the layers of the mesentery, which often shows striation parallel to the intestine. Stercoral ulceration is also present in a greater or less degree.

This distribution of cicatricial strictures does not agree with the statement of Nothnagel that they are, for practical purposes, nearly always due to tubercular or stercoral ulceration, and that very rarely are they produced by syphilitic ulcers, round ulcers of the duodenum, dysenteric and catarrhal follicular ulcerations, and as a result of typhoid ulcers.

(A) *Strictures due to Ulceration.*

(1) *Tubercular Stricture of the Intestine.*

The tubercular infection may involve the mucous membrane, the submucous tissues, or it may attack the muscular and subserous layers in the form

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which is known as chronic hyperplastic tuberculous tumour of the intestine.

Ulceration of the mucous membrane of the ileum is not infrequent in phthsical patients. The process commences in Peyer's patches by the formation of miliary tubercules. These run together and form ragged ulcers with overhanging edges, which tend to spread round the intestine and to become annular. Narrowing of the lumen of the intestine is not uncommon, but it is very seldom that stenosis is produced, because the contents of the small intestine are fluid, and the patient seldom survives long enough for great contraction to occur. In those cases that do live long enough, multiple strictures are often found, even to the number of seven or eight.

When *the tubercle bacillus attacks the submucous laver*, it produces a round-celled infiltration through which giant cells are scattered, and this may occur without any ulceration of the mucous membrane. Should the granulation tissue be replaced by fibrous tissue, diaphragm-like strictures are produced.

A good deal of attention has lately been directed to *chronic hyperplastic tuberculous tumours* of the intestine by Professor Hartman, of Paris, and many others. In this variety of tubercular infection a great quantity of dense fibrous tissue and fat are deposited in the muscular and subserous layers of the intestine, indeed the latter may become half or even three quarters of an inch thick. The lumen of the bowels is reduced to a narrow channel which may be ulcerated or not. The great hyperplasia found in these cases is supposed to be due to an extremely attenuated form of the tubercle bacillus, or to a mixed infection by the bacillus coli. The ileo-cæcal region is the commonest site for these masses of tuberculous fibrous tissue, but they are also found in the small intestine, and Mr. F. S. Kidd has recorded three cases in the sigmoid flexure which have occurred at the London Hospital in the last year or so.

The chief interest about chronic hypertrophic tuberculous tumours is that they have been very frequently mistaken for carcinoma of the intestine, even after excision.

The chief points in diagnosis are the presence of phthisis and of tubercle bacilli in the fæces, although both of these may be absent. The limits of the tumour are, moreover, less definite, and obstruction occurs much later after the mass has been recognised, being delayed as long as a year or more.

In all forms of tuberculous stricture the neighbouring lymphatic glands are involved, and the mass is very adherent to the omentum and neighbouring structures. Bernay has collected 70 cases of tuberculous strictures, of which eight were of the hypertrophic variety. Of the 45 operated on 37 recovered. In the greater number the mass was excised, but in a few the stenosis was excluded by lateral anastomosis.

Nothnagel and Grawitz describe *stercoral ulcers* of the colon as a common cause of simple strictures, and they believe that they frequently lead to carcinoma. I can only state that we have not had such a case at the London Hospital in thirteen years.

Duodenal ulcers are another rare cause of simple intestinal stricture, but the duodenum is so wide and the contents are so fluid that obstruction is very uncommon. Round ulcers of the duodenum, whether they be situated on the anterior or the posterior wall, nearly always produce a contact ulcer opposite, and as each ulcer extends the other follows it, until in many cases they become annular. Should the ulcer heal and cicatrise a stricture is thus produced. Hour-glass contraction of the stomach is brought about in a similar way. An infective round ulcer on the front of the stomach and near the lesser curve produces a contact ulcer opposite. They simultaneously extend until they straddle the lesser curve, and a saddle-back or horse-shoe ulcer is produced. Since the lesser curve is fixed the contraction of the ulcer as it heals drags up the mobile greater curvature near its middle, and produces the hour-glass contraction. To return to the subject of duodenal strictures. A man, æt. 32 years, was under the care of Sir Frederick Treves at the London Hospital in 1896. For two and a half years he had suffered from symptoms of pyloric obstruction, but at the operation these symptoms were found to be due to a simple duodenal stricture. This stricture was dilated by the index finger and the symptoms were relieved, but a month later he died of profuse hæmatemesis. Duodenal ulcers are usually in the first part of the duodenum, and should a stricture result no bile is found in the vomit from the dilated stomach. Sometimes they involve the biliary papilla, and as they contract persistent jaundice is produced.

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Very rarely a stricture is produced by a duodenal ulcer at the duodeno-jejunal flexure, and then the vomit contains a large quantity of bile.

Hochhaus reported three cases of duodenal stricture due to gall-stones, one at either end of the duodenum and one near the middle, but from his account they appear to have been cases of constriction by peritoneal adhesions rather than true strictures. *Syphilic ulcers* are a very rare cause of intestinal stricture, except in the rectum. Only two cases in the small intestine have occurred at the London Hospital in thirteen years. They are more often found in the jejunum, and they are single or multiple. They are produced by either congenital or acquired syphilis, and are the result of breaking-down gummata in the lymphoid tissue of the intestine.

It is a very curious fact that *typhoid ulcers* practically never produce strictures. Only two authentic cases have ever been recorded—those of Klob and Hochenegg. This extraordinary rarity of strictures is said to be explained by typhoid ulcers being in the length of the gut, because when the ulceration is deep and extensive the patients do not survive.

A very remarkable error has crept into the subject of simple strictures. Nearly all text-books state that *dysenteric ulceration* is a common cause of them, yet this practically never occurs.

Woodward clearly demonstrated this fact from the medical statistics of the American War of Secession. Among the United States troops 287,522 cases of dysentery were recorded, and of these 28,451 suffered from chronic dysentery; 9431 died and 3855 of these were chronic cases but not one single stricture was found among them all.

(B) *Traumatic Strictures.*

A stricture may follow resection of gut, whether the ends are united by Murphy's button, by end-to-end suture, or by lateral anastomosis.

In some cases the injury is due to a blow or kick upon the abdomen, or the passage of a cartwheel which partially ruptures the intestine. As the scar contracts obstruction is produced about four months later. I have had two such cases under my care. The first was that of an ostler who was kicked in the abdomen by a horse five months before I saw him. He vomited and collapsed and was laid up in bed for several days. Abdominal colic continued when he was able to get up and he vomited at intervals. When I saw him, in July, 1905, he had attacks of colic and visible contracting coils of small intestine. I opened his abdomen and found an oblique scar across a coil of jejunum which had produced a kink around which the omentum was adherent. I divided the omentum and performed lateral anastomosis between the coils above and below the stricture. He made a good recovery. The second case was not so fortunate. A little girl, æt. 3 years, was run over the abdomen in August, 1905, and although she was profoundly collapsed she recovered. Four months later she was again admitted to the London Hospital with complete intestinal obstruction which had lasted eight days. I opened the abdomen and found the ileum adherent to the right iliac fossa and strictured in two places, whilst a large aperture was present in the mesentery. Whilst I was separating the adhesions the enormously distended intestine above ruptured and I had to fix a Paul's tube in the hole. She did not survive. It would have been better to have left the adhesions alone and to have performed lateral anastomosis.

(C) *Stricture after Strangulated Hernia.*

This is a very rare condition. Sir Frederick Treves collected 11 cases. One of these was double. In nine of the remainder a single annular stricture was present, and in one a tubular contraction an inch and a half long. The great majority are in the ileum, but two of Sir Frederick Treves' cases were in the jejunum. The symptoms of a progressive stenosis of the small intestine arise from a few months to a year or more after a strangulated hernia has been reduced. It is almost certain that the stricture is the result of a localised gangrene of the mucous and submucous layers of the intestine, where it has been strangled by the neck of the sac, and this explains why these strictures are sometimes double, as in the following case which was under the care of my colleague, Mr. F. S. Eve, in 1903. A woman had been operated on for a left strangulated femoral hernia some months before, and was admitted to the London Hospital with the symptoms of a small gut stenosis of six weeks' duration. Two strictures were discovered in the ileum a few inches apart,

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and the mesentery was puckered in a line between them. The entire damaged portion of intestine was resected, but the patient did not survive.

The symptoms and diagnosis of cicatricial strictures are those of other forms of chronic obstruction, but it is well to emphasise the distinction between simple and malignant strictures.

In simple stricture the case is one of long duration, yet there is little cachexia, and in many of the cases a previous history of ulceration, enteritis, strangulated hernia, or some severe trauma may be obtained. Fæcal accumulation is distinguished from simple stricture by the rarity of contracting coils of intestine in the former condition, and by the very different previous history which is obtained in the two conditions. It must, however, always be remembered that fæcal accumulation may lead to annular stercoral ulcers, and as these heal cicatricial strictures may be produced which may in turn become malignant, so that the previous history of long-established constipation may be very misleading as to the condition which is actually present.

The treatment of simple strictures.—The treatment of simple strictures is very similar to that of cancerous strictures, with which we have already dealt fully in a previous lecture.

If distension and an acute attack of obstruction are present when the case comes under treatment, this must be relieved by an enterotomy or colotomy, and the stricture itself must be dealt with at a subsequent operation. In the majority of cases, however, the obstruction is chronic, and a permanent cure may be immediately achieved by excising the stricture, or by what is a very much safer proceeding, doubling the gut upon itself at the stenosis, and uniting the lumen above and below the stricture by a wide lateral anastomosis of at least 2½ in. This operation is simple, easy and safe, and the cure obtained is permanent. The patient puts on many pounds in weight, and returns to an absolutely normal condition. Although these cases are unfortunately so rare they yield the most satisfactory results in the surgery of intestinal obstruction.

Congenital Occlusion and Stenosis of the Intestine.

Congenital ano-rectal stenosis and occlusions are not included in this description.

Incidence.—Seven cases of congenital occlusions and stenosis of the intestine have occurred in 669 consecutive cases of all forms of obstruction at the London Hospital, and 6 further specimens have been brought together in the Museum by Mr. Arthur Keith. Leichtenstern states that the distribution and proportion is as follows: 75 small intestine, 10 colon, 375 at the ano-rectal junction.

Pathology.—The majority of these strictures occur at those points in the intestine where marked developmental changes take place, such as in the duodenum, where the liver and pancreas bud out from the intestine; at the duodenal jejunal flexure, where extensive changes in the mesenteric attachment occur; at the point where the neck of the yolk sac is obliterated in the lower ileum, at the ileo-cæcal valve, at the sigmoid flexure, and at the ano-rectal junction. At intermediate points strictures and septa have been attributed to fœtal peritonitis, and in the small intestine to the maldevelopment of valvulæ conniventes.

The thirteen cases at the London Hospital were distributed as follows: Jejunum 2, connected with Meckel's diverticulum 6, ileo-cæcal 2—and in one of these the colon terminated blindly at the hepatic flexure, upper sigmoid 1, lower sigmoid 2. The degree of stricture varies widely. The lumen of the bowel may be reduced for a variable distance and to a variable degree so that a tubular stricture is produced. The walls of this narrowed portion may be normal in structure. Such a case at the recto-sigmoid junction 1½ in. long was under the care of my colleague, Mr. Hugh Rigby. In other cases a narrow permeable stricture is present or a perforated septum. In yet others a complete septum occurs or a narrow impermeable stricture. Finally, the gut may be interrupted for a longer or shorter distance, and the blind ends may be united by a fibrous cord or may be quite independent.

The conditions found in the above thirteen cases were: tubular stricture 1, permeable stricture 4, impermeable stricture 4, complete septum 2, interruption 2. Congential strictures are frequently multiple. Treves has recorded a case in which six existed, and Schüppel one in which no less that ten were present in the small intestine. Four examples of double strictures occurred in my series of thirteen cases: (1) Two septa at the middle of

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the jejunum with one inch of gut between them (Turner); (2) two stenoses, one above and one below a Meckel's diverticulum; (3) stenosis of the end of the ileum and at the cæco-colic junction; (4) occlusion of the ascending colon by the upper cusp of the ileo-cæcal valve and interruption of the colon at the hepatic flexure.

Congenital strictures of the duodenum are usually in the second part and just above the opening of the bile-duct, but they may be situated below that point. The strictures in connection with Meckel's diverticulum are more often above than below that structure.

Septa, when they cause no external constriction of the gut, are composed of the mucous membrane and the circular muscular coat, but do not involve the longitudinal fibres.

Symptoms.—When the stricture is situated in the small intestine and is permeable, or when a perforated septum is present, a truly remarkable degree of constriction may exist without producing any symptoms. In such cases the patient may reach to adult life, and the stricture may be found accidentally at an autopsy (Hudson and Rolleston). In other cases symptoms of chronic stenosis gradually appear (Hudson), or acute intestinal obstruction may be precipitated by a foreign body lodging in the stricture or a volvulus occurring in connection with it, as happened to a case under the care of Sir Frederick Treves at the London Hospital in 1894, in which a volvulus and a congenital stricture were found at the autopsy on a man æt. 36 years.

When the intestine is occluded the baby does not as a rule survive more than five days. During the first day little is noticed. On the second day the absence of stools attracts attention, and the child frequently vomits. In two of the cases at the London Hospital the bowels were open and mucus was passed, in one case mixed with blood.

On the third day, in those cases in which the obstruction is in the lower part of the intestines, the abdomen becomes distended and coils of intestine may be seen to contract at intervals.

When the occlusion is situated in the duodenum the stomach is obviously dilated, and may be seen to contract. During these contractions the screams of the infant indicate that it is suffering from colic. The vomited matter does not contain bile when the stricture is above the diverticulum of Vater, but when the occlusion is just below that structure or at the duodenal-jejunal flexure, bile is present in the vomitus.

The baby rapidly emaciates and becomes exhausted and collapsed, and, as has been said, dies about the fifth day.

In those cases in which the stricture is permeable but sufficiently narrow to cause obstruction, an inadequate quantity of meconium is passed by the rectum, but distension, contracting coils of intestine, colic and vomiting are present, although these signs appear later and are less severe than in occlusion. In such cases the infant may live for a variable period which may extend to years, as in those cases of congenital dilatation of the colon which are due to a tubular contraction of the colon.

The prognosis is absolutely bad. No case of occlusion has survived at the London Hospital.

Treatment.—It is doubtful if any treatment is of much avail. The intestine has been opened many times above the obstruction, but the operation appears to only hasten the inevitable ending. Colotomy is probably worth doing where the obstruction is in the lower part of the colon.

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April 6th, 1908.

We have received from Burroughs Wellcome & Co. samples of their "Tabloid" Phenol and Menthol Compound; this will be found a pleasant and effective means of administering a gastric stimulant and antiseptic. The combination is useful to promote appetite and digestion, to relieve pain in dyspepsia, and to prevent or arrest fermentation in dilated stomach.

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